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LACTOSURIA AND AMINO-ACIDURIA IN INFANCY

A CASE REPORT

H. DE V. HEESE, M.D., B.Sc., M.R.C.P. (EDIN.), D.C.H. and G. M. POTGIETER, M.B., CH.B., M.MED. (PATH.)

Departments of Child Health and Chemical Pathology, University of Cape Town, Observatory, Cape

Several reports describing carbohydrate intolerance in infancy as a result of intestinal carbohydrase deficiencies have recently appeared. Darling *et al.*[1] reported the finding of considerable lactosuria and amino-aciduria in two related infants who presented with failure to thrive, diarrhoea and vomiting. The condition proved fatal in both cases. Holzel *et al.*[2] described a similar clinical presentation in two siblings. Diarrhoea and failure to thrive while on milk were the main signs. On a lactose-free diet both infants improved and progress was maintained. Lactose-tolerance tests confirmed the inability to metabolize this sugar. No reference was made to amino-aciduria or lactosuria. Further, Weijers *et al.*,[3] in a preliminary communication, reported the investigation of patients suffering from invertase and/or maltase deficiencies. They had also seen cases of lactase deficiency. Again, no reference was made to amino-aciduria or the finding of sugars in the urine. Inall[4] reported that both sucrose and lactose appeared in the urine while the patient was receiving these sugars. Improvement followed the introduction of lactose- and sucrose-free diets accompanied by the disappearance of these sugars from the urine. No amino-aciduria was present.

The basic biochemical lesion in all these cases appeared to be an absence or deficiency of the particular carbohydrate-splitting enzyme in the intestine. The ingested disaccharide could therefore not be split into its monosaccharide components. This resulted in abnormal fermentation in the bowel from bacterial action on the sugars with the production of large quantities of lactic acid and other volatile fatty acids.[3] These substances produce bowel irritation, increased peristalsis and diarrhoea. Some of the unsplit sugar may be absorbed, but because it cannot be utilized it is rapidly excreted in the urine.

A patient resembling those described by Darling *et al.*,[1] with lactosuria and amino-aciduria, was admitted to Groote Schuur Hospital, Cape Town, recently. The lactosuria was, however, not recognized initially, through failure to examine the urine by a reduction method in addition to methods specific for glucose ('tes-tape').

CASE REPORT

A European girl, aged 2 months, was admitted to the paediatric ward under the care of one of us (H. de V. H.). The infant was delivered at home, weighing 5 lb. 8 oz., after a gestation period of 37 weeks. The immediate neonatal period was apparently uneventful except for failure to gain weight normally. Breast feeding was stopped after 2 weeks and was followed by artificial feeding with full-cream milk. Loose green stools and vomiting began at the age of 1 month. No relevant family history was obtained from the father, aged 27 years, and the mother, aged 24 years. Two siblings, a boy aged 3 years and a girl aged 2 years, were healthy.

On admission, the infant weighed 6 lb. 6 oz. and was moderately dehydrated, with pallor of the mucous membranes and a scaly rash on the trunk. Clinical examination showed no other abnormalities and no changes were noted in the corneas, lenses, anterior or posterior chambers, and retinae of the eyes.

Blood haemoglobin concentration was 9·5G. per 100 ml. and white blood count 22,000 per c.mm. Urine was acid to litmus paper, contained a trace of protein but no glucose (tes-tape), ketones, bilirubin or urobilin, and there was an essentially normal spun deposit.

After a provisional diagnosis of gastro-enteritis had been made, therapy with intravenous half-strength Darrow's solution, containing 2·5% dextrose, was begun. In addition, the infant received antibiotics. Intravenous therapy was discontinued after 24 hours and was followed by half-strength Darrow's solution, 5% dextrose and, subsequently, oral skimmed milk. After 4 days on this régime, clinical improvement was evident, accompanied by a gain in weight of 11 oz. The feed was now changed to half-cream milk. Shortly afterwards diarrhoea re-appeared with rapid clinical deterioration and dehydration. The infant remained apyrexial and repeated stool cultures, blood culture and cerebrospinal-fluid examination yielded negative results.

Some improvement followed further intravenous therapy. On the tenth day after admission, a reducing substance yielding a brick-red deposit on testing by Benedict's qualitative method was detected in the urine. This was confirmed on subsequent testing. The urine had been tested several times previously but only with tes-tape (glucose-oxidase). Blood-sugar levels at the time of the Benedict tests were 43 and 62 mg. per 100 ml. (Hagedorn and Jensen).

Further episodes of diarrhoea and rapid dehydration warranted the administration of hydrocortisone intravenously. Acidosis (serum bicarbonate of 12·5 mEq. per litre) was treated satisfactorily with a solution containing 30% potassium citrate and 30% sodium citrate. A blood transfusion corrected the anaemia. Further fluid requirements were guided by frequent serum-electrolyte determinations. However, in spite of temporary remissions while on intravenous therapy, relapses regularly recurred on oral feeding with milk, since the significance of the positive Benedict's test was not appreciated early, and the infant died 25 days after admission, before a definite diagnosis could be made.

Results of additional special investigations were: serum albumin 2·1G. per 100 ml., globulin 1·4G. per 100 ml., serum cholesterol 47 mg. per 100 ml., inorganic phosphorus 3·4 mg. per 100 ml., alkaline phosphatase 5 units per ml. (Bodansky). X-ray examination of chest, wrists and abdomen was normal. Wassermann and Berger reactions were negative.

Special Methods

A 24-hour specimen of urine was submitted late in the illness for identification of the reducing substance in the urine and for testing the presence of amino-aciduria.

The reducing substance was identified by one-dimensional chromatography, using a solution containing xylose, fructose, glucose, sucrose, lactose and galactose as reference markers. In 2 different solvent systems—isopropanol-water, 160:40, and n-propanol-ethyl acetate-water, 140:20:40—the R_F* of the unknown corresponded exactly to that of lactose. The aniline diphenylamine reagent was used for locating the sugars.

For amino-acid chromatography an aliquot of the urine was

* R_F (ratio of flow) represents the distance the material has moved from the original point of application divided by the distance the solvent front has travelled from that point.

desalted in an ion-exchange column containing zeocarb 225 in the hydrogen cycle followed by elution with concentrated ammonia.[5]

Two-dimensional paper chromatography was performed on Whatman's No. 1 paper using as first solvent butanol-acetic acid-water, 120:30:50, followed by t-butanol-methyl ethyl ketone-ammonia (S.G. 0·880)-water, 40:40:10:20. Ninhydrin, 0·2% in acetone, was used as location reagent. This showed a generalized amino-aciduria (Fig. 1).

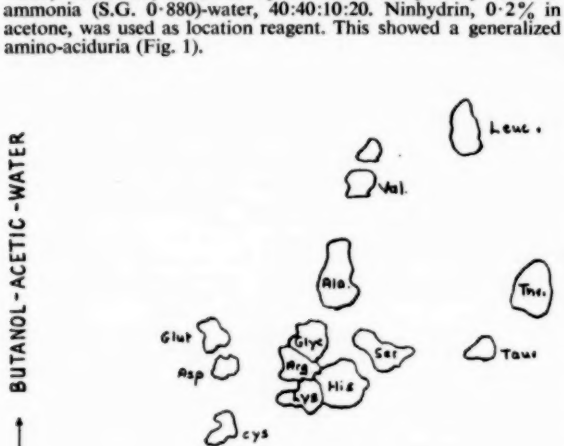

Fig. 1: Tracing of original amino-acid chromatogram. Glut. = glutamic acid, Asp. = aspartic acid, Cys. = cystine, Lys. = lysine, Arg. = arginine, Glyc. = glycine, Ala. = alanine, Ser. = serine, Taur. = taurine, Thr. = threonine, Leuc. = leucine, Val. = valine, and His. = histidine.

Summary of Autopsy Findings

The body was that of an oedematous female infant weighing 2,740 grams. Apart from focal areas of collapse in both lungs and a moderate degree of fatty change in the liver, the only significant abnormalities were confined to the rectum. Four small ulcers, 1 - 2 mm. in diameter, were present in the lower rectum. The intervening mucosa was normal. The stomach, duodenum and proximal jejunum were free of lesions. The rest of the bowel was accidentally discarded before it could be examined in detail, but on preliminary inspection no obvious abnormalities were noted.

Histologically the lesions in the rectum appeared to be acute submucosal abscesses. Definite ulceration could not be demonstrated in the sections examined. The exact nature of these lesions could not be determined and postmortem bacteriological examination failed to reveal any pathogens.

DISCUSSION

Although the delay in detecting the reducing substance in the urine of this infant prevented complete biochemical investigation, the clinical presentation associated with amino-aciduria and lactosuria suggests that she suffered from a condition similar to that described by Darling *et al.*[1] The rapid deterioration each time a change was made to a milk-containing feed, with remissions while on intravenous therapy, lends further support.

Small quantities of lactose are commonly found in the urine of newborn and especially premature infants. The amount excreted is usually less than 100 mg. per 100 ml. and is often detected by chromatographic methods only.[6] This is thought to be due to inadequate lactase activity, because this enzyme is the last of the disaccharidases to appear and may not be present until the eighth foetal month.[7] For this reason the mere finding of traces of lactose in the urine of newborn children is not sufficient evidence for diagnosing this syndrome. Similarly, amino-aciduria may occur in the premature and newborn infant. Although quantitative urinary lactose estimation was not performed in this case, the brick-red deposit constantly obtained on qualitative testing with Benedict's reagent indicated a urinary concentration of at least 2 G. per 100 ml. This finding in the presence of blood-sugar levels of only 43 and 62 mg. per 100 ml. does indicate significant lactose intolerance.

Neither Holzel *et al.*[2] nor Weijers *et al.*[3] referred to urinary findings in their cases. The incidence of amino-aciduria and the frequency with which the offending sugar occurs in the urine can therefore not be assessed.

A hereditary basis seems likely since the condition has been found in siblings.[1,2] The father, mother and siblings of the present patient were investigated for lactosuria with negative results. It has not yet been established whether the condition is a permanent defect or just delayed maturation of the enzyme system. Holzel *et al.*[2] stated that their patients were perfectly well at the ages of 15 months and 10 years respectively, but made no reference to lactose tolerances at those ages. This may therefore be a temporary inability to metabolize these disaccharides efficiently, and speculations as to possible relationships between carbohydrate and amino-acid anomalies would appear untimely at this stage.

The importance of recognizing the condition and removing the offending sugar from the diet is obvious, because in addition to producing diarrhoea, hypoglycaemia may be a serious complication due to inability to metabolize the available carbohydrate.

Weijers *et al.*[3] found significantly raised levels of lactic acid and volatile fatty acids in the stools of their patients. If the faecal lactic excretion exceeds 1 gram in 48 hours, carbohydrate intolerance should be considered as a probable cause. Suitable carbohydrate-tolerance tests employing the various disaccharides will confirm the diagnosis and determine which enzyme is deficient.

The danger of omitting to test urine by a reduction method in addition to methods specific for glucose (glucose-oxidase) is well illustrated by this case, since an important diagnostic clue was overlooked at first.

SUMMARY

A fatal case of amino-aciduria and lactosuria is described and the findings in other reported cases presenting with similar syndromes are briefly reviewed.

The importance of employing a reduction test, in addition to a glucose-specific method, as a screening test for the presence of urinary sugar in early infancy is stressed, particularly in view of the possibility of successful therapy being instituted on detecting such an abnormality.

We wish to record our thanks to Dr. S. Katz and Sister N. Duke for their assistance; Dr. H. A. van C. de Groot for the postmortem findings; Prof. F. J. Ford and Prof. J. E. Kench for their interest; Prof. J. G. Thomson for making the postmortem findings available to us; and Dr. J. G. Burger, Superintendent of Groote Schuur Hospital, for permission to publish this case report.

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EDITORIAL : VAN DIE REDAKSIE

THE HAZARDS OF RADIATION

In the past decade the problems posed by radiation hazards have received more and more attention, particularly with the increasing amount of radiation produced by medical and other sources. Numerous papers and other publications have appeared of which two, published by the Medical Research Council in 1956 and 1960 and entitled *Hazards to man of nuclear and allied radiations*[1] and *Radiological hazards to patients*[2] respectively, are extremely detailed and authoritative. Many questions that were previously the subjects of much speculation are now more definitely answerable on a far surer basis of fact.

There have been notable changes of thought. Formerly it was believed that an individual, exposed to a high dose of radiation, carried for his lifetime thereafter a heightened risk of leukaemia. Now, however, from observations at Hiroshima and Nagasaki and observations on those irradiated in the treatment of ankylosing spondylitis, it can be stated with some confidence that the risk of leukaemia following radiation will decline after the lapse of a decade. It was also thought that the biological effects of a given dose of irradiation were similar, whether given rapidly or slowly. Recent work suggests that a dose given over a long period has less effect than the same 'acute' dose, and this work is supported by earlier findings that genetic damage is related to dose rate.[3]

This change of opinion, therefore, puts far more emphasis on the sudden doses of medical radiology and less on the slower exposure to background and fall-out radiation. Although the subject seems to be highly technical, there are a number of simple facts which amply illustrate the important points, and it appears to be important at this time for the medical profession as a whole to give a great deal of thought to this problem which may affect future generations more than is universally recognized.

1. *The Genetic Hazards of Medical X-rays*

Muller[5,6] in 1950 and 1954 pointed out the danger of irradiating the gonads either of the adult or of the foetus *in utero*. He stated that small doses of irradiation may not harm the foetus directly and yet may produce gene mutations in the foetal gonads which could cause a great deal of harm to subsequent generations. The mutants thus produced are unfortunately nearly all harmful and the effect on the individual usually so slight as to be impossible to detect. Nevertheless, irradiation of the gonads may be detrimental by causing slight undetectable changes in the offspring which, by being passed on to subsequent generations, may eventually be lethal. Also Russell and Russell[7] showed that developmental abnormalities can be produced in the embryos of mammals by irradiation. Most of the gross abnormalities were produced at a time of development which corresponded with the second to the sixth week of human gestation.

The calculated total genetic dose from medical X-rays is 19·3 milliroentgen annually per person, compared with the inescapable dose from background radiation of 85 - 106 milliroentgen, and the gonad dose from fall-out, which is a measure of the genetic consequence of nuclear explosion, of 1·2 milliroentgen per year.

(*a*) *Medical diagnostic radiology*. This source contributes an annual average of 14 milliroentgen per person which is a lower estimate than was made in 1956.[4]

The gonad dose was found to be not excessive and similar to values previously reported for many examinations. It was lower than earlier figures for pelvimetry and salpingography, possibly as a result of the publicity given to these procedures recently. However, owing (mainly) to poor technique, other examinations such as chest radiography showed a high average. Thus, the testis was in the primary beam for 10% of all chest X-rays and the ovary similarly involved in 51% of such X-rays—a very high figure indeed.

Radiology of the chest makes up 5% of the genetic dose from diagnostic radiology. Obstetric radiology contributes one third of the total, and irradiation of the pelvis and femur a further third of the total. Mass miniature X-rays of the chest and dental X-rays contribute only one thousandth part of the annual genetic dose. From these figures it is obvious that the use of simple measures would reduce the gonad dose very considerably.

(*b*) *Radiotherapy* contributes a smaller annual average dose, 5·3 milliroentgen per person, of which only 10% is attributable to the treatment of malignant disease. More than 50% of the total radiotherapy dose comes from treatment of skin conditions and most of the remainder from the treatment of ankylosing spondylitis. Only a very small proportion of the total can be attributed to radioactive isotopes used in diagnosis and treatment.

Two striking and important points are evident from the dose survey: (1) The gonad dose is not reduced in diagnostic radiology by making the beam of an X-ray more penetrating (an optimum is reached at about 70 KV. with 1 millimetre of aluminium filter), and (2) through faulty techniques a large and totally unnecessary genetic dose can be contributed. The same applies to radiotherapy for non-malignant conditions.

Pregnancy in itself poses many particular problems and special precautions should be adopted for pregnant women. These precautions are necessary for every female of childbearing age who might be pregnant, because the embryo often attains its highest degree of radiosensitivity (somatically) before the mother's pregnancy is suspected. However, at any stage of pregnancy irradiation of the mother's abdomen involves irradiation of the foetus and this is a double genetic dose. At present one quarter of the population's annual genetic dose arises from irradiation of the foetus.

For very similar reasons, extensive or repeated X-ray examinations of children and adolescents need to be specially

considered and selected. It is estimated that the total genetic dose can be reduced to about 6 milliroentgen by the adoption everywhere of careful techniques giving low gonadal exposure. Larsson[8] makes the same point in his assessment of the genetic dose in Sweden.

2. *Leukaemia and Radiation*

After the initial report by Stewart *et al.*[9] that acute leukaemia in childhood was much more likely if the mother had been subjected to much X-ray exposure in pregnancy (e.g. pelvimetry), and the demonstration by Court Brown and Abbatt[10] that there was an increased incidence of leukaemia in patients with ankylosing spondylitis treated with deep X-ray therapy, a great deal of thought and research all over the world has been stimulated.

(*a*) *Radiology*. On the whole the danger of maternal X-radiation to the foetus has not been substantiated in so far as the incidence of leukaemia is concerned. Court Brown, Dall, and Bradford Hill[11] showed no increased incidence of leukaemia in 39,166 foetuses of mothers who were known to have had abdominal or pelvic diagnostic X-rays, and this proved true even in 750 women where the X-rays were taken during the first 3 months of pregnancy. In their review of five other surveys conducted by Kjeldsberg,[12] Kaplan,[13] Polhemus and Koch,[14] Ford *et al.*[15] and MacMahon,[16] they point out that the evidence is conflicting. In three of these surveys a greater proportion of mothers of leukaemic children had been irradiated during pregnancy than among mothers of the control children; in one there was no difference and in one the opposite was true. They conclude that 'there is no evidence of any disproportionate occurrence of leukaemia among the children most heavily irradiated, nor among the children who have been irradiated early in intrauterine life'. Lewis,[17] who reviewed the records of Queen Charlotte's Hospital in London, reached similar conclusions.

(*b*) *Exposure to radioactive isotopes*. This is another way in which radiation may cause leukaemia. Radioactive gold and yttrium have been used in the treatment of tumours. Radio-iodine (^{131}I) has been used in large numbers of patients with thyrotoxicosis, and radiophosphorus (^{32}P) has been used in polycythaemia vera.

Pochin, in a review of 18 cases of leukaemia, suggests that there is no evidence at present that radio-iodine treatment is inducing leukaemia. In spite of the work of Wasserman,[18] Schwartz and Ehrlich,[19] and Lawrence,[20] no firm conclusion can be reached at present about the relative incidence of leukaemia in polycythaemic patients who have been irradiated.

(*c*) *Radioactive traces*. This is another source of exposure to radiation. Radioactive chromium (^{51}Cr) is used in blood-volume estimations and in studies on the survival of red cells, radioactive iron (59 Fe) is used in studies on iron metabolism, and radiocobalt (60 Co) is used experimentally in studies on the metabolism of Vitamin B_{12}, to mention only a few which are being used increasingly in research and diagnosis. Although the dose for one estimation may be carefully adjusted, the cumulative dose may be at fault if not carefully watched. The somatic effect of these isotopes is the subject of intensive investigation at present.

What then are the main conclusions to be drawn at the present time from the facts at our disposal?

As regards the genetic hazard of medical X-rays, it would appear from the second report of the Adrian committee on *Radiological hazards to patients*, that there is no need for major restriction in radiological practice, provided that the proper precautions are taken in all instances. This places the responsibility for ensuring that no unnecessary gonadal irradiation occurs on the person requesting the X-ray and on the radiologist and his staff.

Concerning the question of radiation and leukaemia, no cast-iron opinion can be expressed at present. The scientific evidence is still conflicting and it is possible only to attempt a judicious opinion. The important question to be answered is: which will lead to greater human suffering—using or withholding diagnostic abdominal X-rays in pregnant women? This answer will not be available until obstetricians provide data to show to what extent information provided by X-rays has led to reduction in maternal and foetal morbidity and mortality. It does however seem that radiation is a definite danger to the embryo in the first trimester and should always be avoided when possible.

Every member of the medical profession should read the recommendations in the report of the Adrian Committee and do all in his or her power to implement these recommendations in everyday practice.

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PUBLIKASIE VAN DIE APTEKERSWOORDEBOEK

'n Tweetalige Engels-Afrikaanse en Afrikaans-Engelse Aptekerswoordeboek het so pas verskyn. Hierdie woordeboek is bewerk deur 'n verteenwoordigende komitee van die Vaktaalkommissie en sy Sekretariaat in die Vaktaalburo van die Suid-Afrikaanse Akademie vir Wetenskap en Kuns, met die *Voorlopige lys van aptekersterme* van die Akademie as grondslag. Die uitgewers van die woordeboek is die Suid-Afrikaanse Akademie van Wetenskap en Kuns. Dit word versprei deur die Nasionale Boekhandel Beperk, Parow, Port Elizabeth, Bloemfontein, en Johannesburg. Prys: R1.25, posgeld 3c ekstra.

Die verskyning van 'n woordeboek soos hierdie is belangrik uit baie oogpunte. In die eerste plek is dit 'n verdere nuwe mylpaal wat bereik is deur die Afrikaanse vaktaal.

Verder is dit, soos die voorsitter van die vaktaalkommissie in sy voorwoord sê, nie 'n maklike taak om die terminologie van 'n jong en groeiende taal te koördineer nie. Dit is dus wel 'n stap vooruit dat dit moontlik is om die Afrikaanse vakterminologie op landswye basis in so 'n mate te koördineer dat daar nie meer onnodige uiteenlopendheid en misverstand ondervind kan word nie.

Hierdie woordeboek is die vrug van die arbeid van die verteenwoordigende komitee vir die aptekerswoordeboek waarin daar verteenwoordigers dien van die Vaktaalburo, die Suid-Afrikaanse Aptekerskommissie en Aptekersvereniging, die W.N.N.R., die Taaldiensburo, die Witwatersrandse Tegniese Kollege, die Suid-Afrikaanse Spoorweë, die Departement van Onderwys, Kuns en Wetenskap, die aptekers van die Pretoriase Algemene Hospitaal, en lede van die Universiteite van Stellenbosch, Pretoria, Witwatersrand, en Potchefstroom. By die bewerking van die manuskrip in sy finale vorm het ook nog ander belangrike liggame en persone 'n rol gespeel; onder andere het die Taalkomitee van die Universiteit van Stellenbosch, byvoorbeeld, die hele manuskrip deurgewerk en aanbevelings gemaak. Die Aptekerswoordeboek is dus in die ware sin van die woord 'n nasionale kulturele bydrae.

Die verskyning van die woordeboek is ook 'n treffende bewys van die belangrike plek wat die aptekerswese al in ons land ingeneem het. Mediese dienste het in die afgelope aantal jare al meer ontwikkel in gesamentlike dienste. Die geneesheer staan nie meer alleen, soos dit in die verlede die geval was, in die stryd teen siekte en swak gesondheid nie. Hy tree al meer op as leier onder die gelykes van 'n span waarin vervaardigers van farmaseutiese produkte, en aptekers, verpleegsters, fisioterapeute, röntgenterapeute, arbeidterapeute, maatskaplike werkers, en baie ander almal belangrike skakels is. In ons ontwikkelende vaktaal moet die taal en terminologie van die geneesheer self, sowel as van al hierdie groepe persone, weerspieël word.

Daar wag dus nog 'n groot taak vir ons om in die toekoms aan te pak. In die tussentyd wil ons graag alles in ons vermoë doen om die reeds bereikte prestasies te help bestendig. Om hierdie rede wil ons dus graag die algemene gebruik van die *Aptekerswoordeboek* sterk aanbeveel by geneeshere, mediese studente, verpleegsters, vervaardigers van farmaseutiese produkte, aptekers, en al die ander lede van die ‚grotere mediese professie' wat ons hierbo genoem het.

ARTERIAL INJURIES

L. GECELTER, M.B., B.Ch. (RAND), *Department of Surgery, University of the Witwatersrand*

An analysis of 50 cases of major arterial injury was made in patients presenting for treatment at Baragwanath Hospital over the past 2½ years. Those patients who died before any form of therapy had been carried out were excluded from the series, as were those with injuries involving minor arteries such as the radial, ulnar, superficial temporal, posterior tibial and dorsalis pedis, which commonly occurred.

The location of the arterial injuries are summarized in Table I. The commonest vessels involved were the brachial, femoral, carotid, and subclavians; arterial injuries being dispersed throughout the arterial tree.

TABLE I. LOCATION OF ARTERIAL INJURIES

Aorta	Thoracic	2
	Abdominal	1
Carotid	Common	7
	Internal	0
Vertebral		2
Subclavian		3
Axillary		6
Brachial	Above profunda	3
	Below profunda	7
Femoral		10
Popliteal		5
Pulmonary		1
Anterior tibial		2
Transverse cervical		1
Total		50

Two cases of injury of the anterior tibial artery were included in the series because of complications — one developed gas gangrene and the other an aneurysm which resulted in severe anoxia of the extensor compartment. Both required extensive fasciotomy and excision of the extensor muscles of the leg.

Arterial injuries were classified according to the usual 4 types encountered, lacerations accounting for 70% of the lesions (Table II). Transections only occurred in 14%.

TABLE II. TYPES OF ARTERIAL INJURIES

1.	Transections	14%
2.	Lacerations (including intimal dissections)	70%
3.	Contusion (with thrombosis)	4%
4.	Spasm — Benign	6%
	Spasm — Malignant	6%

In most series the proportion of lacerations to transections is approximately equal and in De Bakey and Morris's[1] series of 136 cases the proportion was 41 : 52%.

In the present series the results could not be correlated with the type of arterial injury. If any, the results were slightly better with complete transections than with incomplete lacerations.

No cases of contusion occurred without associated thrombosis. Spasm was classified as benign or malignant according to the type of injury sustained, the response to treatment, and the final result. Kinmonth[2] distinguishes a benign local or myogenic spasm as distinct from a more extensive circumferential neurogenic spasm which involves collaterals due to severe crushing or an occluding injury, as may occur with a fracture or a tourniquet-occlusion.

Three patients with benign spasm presented with brachial artery occlusion and all responded to open operation and application of papaverine, 2·5% locally; whereas in 3 patients with extensive spasm in the popliteal region, resulting from crushing injury, bullet concussional effect, or fracture-dislocation of the knee, revascularization failed in the lower extremity, and ended in amputation. A dissection of one of the limbs subsequently revealed

thrombosis of the anterior and posterior tibial and peroneal arteries distal to the site of spasm, as well as thrombosis of the popliteal veins. It would appear that prolonged spasm may precipitate thrombosis in the distal static vessels.

Intimal dissection was recorded in only 1 case and was caused by acute angulation at the site of a closed midshaft fracture of a femur. It may also occur in association with concussional effects of a high-velocity missile or in a closed injury with bruising of an artery.

With rupture of a subintimal clot, arterial pressure causes a dissection of the subintimal space which may spread in the wall of the artery for a considerable distance.

AGE

The age variation was between 9 and 43 years. One patient was 60 years old. The median age was 24 years. All but 4 were male subjects and the problem of atherosclerotic vessels did not occur in any of the patients.

TIME OF PRESENTATION AFTER INJURY

No correlation could be found between the time of presentation at hospital after the injury and the end results. Good results were obtained in patients operated on 24 hours after injury, and the reverse also applied. Delay did not contraindicate surgery.

Hughes[3] stresses the patient's general condition, amount of shock, exposure, collateral supply present, soft-tissue injury, and the affected artery, and uses these criteria as a guide rather than the time factor. If there is any doubt regarding the viability of the limb it is better to repair the artery, which may salvage a part of the limb. In civilian practice arterial injuries present earlier and are treated sooner than during war time.[3] The patients in this series presented in two main groups:

1. *Within 24 hours*
   - (i) Within 6 hours, 31.
   - (ii) Between 6 - 12 hours, 4.
   - (iii) Between 12 - 24 hours, 1.

   Total 36 cases.
2. *After 24 hours*

   Variation between 2 days and 4 years. Median 1 month. All these patients presented with aneurysms or fistulae. Total 14 cases.

ASSOCIATED INJURIES

These are important in arterial injuries and occurred in the present series on 29 occasions (58%). They included the following: (1) Bone or joint injury 6, (2) extensive soft-tissue injury 3, (3) nerve injury 8, (4) major vein injury 8, and, (5) unassociated injuries 4.

1. *Bone Injury*

In 4 cases of arterial injury fracture of the femur occurred — the arteries sustaining transection, spasm, subintimal dissection, and mechanical obstruction due to bone fragment, respectively.

One dislocated knee caused extensive contusion with thrombosis, and 1 supracondylar fracture of the humerus resulted in benign spasm.

2. *Extensive Soft-tissue Injury*

This was an important factor in the thigh only, where it occurred on 3 separate occasions in open injuries. This resulted in extensive muscular collateral involvement. Collaterals are better developed in a muscular region such as the thigh than in a non-muscular part like the knee, although there are more named collaterals in the knee.[4]

Extensive soft-tissue injury associated with arterial injury, therefore, has a bad prognosis.[5] Of the above 3 cases of extensive soft-tissue injury, 1 required an above-knee amputation and the remaining 2 were left with permanently ischaemic limbs.

3. *Nerve Injuries*

An associated nerve injury should be assessed in detail at the original examination with a view to differentiating it from ischaemic neuropathy. An associated nerve injury will present initially, not be progressive, and will conform with typical motor and sensory topography.

Ischaemic neuropathy is progressive; it is characterized by a typical glove or stocking distribution, selective sensory loss and delayed pain sensation which is the last to disappear. Epicritic sensation is involved early. Wrist drop and weakness of the long extensor to the big toe is a notable feature of ischaemic neuropathy. Intrinsic muscles of the foot and hand are also involved.[4]

In this series nerve lesions occurred in the upper limb and root of the neck only, with partial involvement of the brachial plexus (2), Horner's syndrome (2), median nerve (3), and musculocutaneous nerve (3). No nerve injuries occurred in the lower limbs where the arteries and nerves are at some distance from each other.

In one patient with a wrist drop and intrinsic muscle palsy following brachial artery occlusion, the radial nerve was explored and found to be normal in appearance — a case of ischaemic neuropathy.

All the nerves were repaired with the arteries at the original procedure.

4. *Vein Injuries*

All veins other than the superior vena cava and inferior vena cava above the renal veins may be ligated, but the tendency is for repair rather than ligation. Treatment will depend on the site, collateral supply, type, and accessibility of the injury and general condition of the patient. Deep-seated venous injuries such as occur in the root of the neck and abdomen may be more difficult to treat than arterial injuries. Often repair by lateral suture may be easier than ligation. An unsuccessful repair may result in thrombosis which may become propagated.

In the present series 8 major venous injuries occurred: 3 internal jugular veins — all ligated, 1 axillary vein — ligated, 1 brachial vena comes — ligated, 2 femoral veins — 1 ligated and 1 repaired. No complications, attributed to these injuries, occurred.

The 8th case was a contused popliteal vein with intimal clot which occurred as a result of missile concussion; the clot was soft and friable and small emboli had probably occurred.

5. *Unassociated Injuries*

These lower the patient's general resistance and may

mask the arterial injury. This occurred in the following circumstances:

(a) Head injury — semicoma; closed abdominal injury, as a result of being run over by a car. A crushed contused popliteal artery resulted in an above-knee amputation.

(*b*) Haemothorax — on 3 occasions; patients being treated conservatively for haemothorax, in 2 cases the condition was caused by a lacerated subclavian artery and in 1 by a lacerated aorta.

DIAGNOSIS ON ADMISSION

A study was made of the provisional diagnosis by casualty officers and house surgeons on arrival of the patients at the wards. In only 32 patients was a diagnosis of arterial injury or a condition indicating arterial injury made. A provisional diagnosis of 'stab neck', 'haemothorax', 'lacerated thigh', 'multiple injuries with shock', or any urgent direct admission was allowed. In the remaining 18 cases arterial injury was misdiagnosed for 3 main reasons:

1. *Aneurysm, called an abscess,* occurred in 6 cases: 4 brachial aneurysms, 1 axillary aneurysm, and 1 vertebral aneurysm diagnosed as tuberculous adenitis.

Two patients were booked for surgery for incision of an abscess. One was incised and one was needled.

This mistake is not difficult to make. An aneurysm need not pulsate or have a bruit. Blood in the extravascular compartment causes general and local effects of inflammation and may closely resemble an abscess. Occasionally an infected pulsating haematoma occurs.

2. *Overlooked because of associated injuries:* 1 patient in semicoma had closed abdominal injuries (popliteal artery occlusion was only detected 48 hours after laparotomy) and nerve injuries occurred in 3 patients in whom brachial artery occlusion was not diagnosed — the patients having been admitted with radial, median nerve, or partial brachial plexus injuries.

3. *In fractures and dislocations:* 5 patients with a fractured femur and 1 with a dislocated knee were admitted without associated arterial injury being diagnosed, although in all cases the pulse was not present below the fracture. Two patients were transferred from other hospitals and all were radiographed before admission. No provisional splints had been removed in those patients transferred from other centres. House surgeons in the wards also overlooked arterial injuries.

TREATMENT

Blood Replacement

The amount of blood replaced varied between nil and 14 pints. The average was 3·1 pints and the median was 4 pints. The requirements were greater in acute injuries than in aneurysms and fistulae, and were greater in arterial lacerations and transections. More replacement was required where bleeding was difficult to control, as in the neck, axilla, and thorax.

More than 3 pints of blood were seldom required in injuries of the arm and thigh where fracture or extensive soft-tissue injury did not occur.

In arterial surgery, the aim of replacement should be to maintain an adequate blood pressure throughout the procedure particularly after reconstruction of the vessel, where a drop in blood pressure and circulatory stagnation may result in clotting at the site of repair. Inadequate fluid replacement may manifest itself by a fall in blood pressure during the induction stage of anaesthesia. Overloading of the circulation may occasionally occur with the use of hypothermia and in the repair of arteriovenous fistulae.

Exposure

Before treatment of the affected artery, the exposure should allow tape control of the artery both proximally and distally. This should be done in all cases. Peep-hole surgery may result in tragedy and occurs particularly in the root of the neck. There should be no hesitation in removing wide segments of clavicle and costal cartilages, and splitting the manubrium longitudinally with lateral extension, to secure adequate exposure.[16]

The upper carotid and vertebral systems may be exposed by an oblique or transverse incision in the neck.

Arterial injuries around and including the heart may be adequately exposed by an anterior transverse intercostal incision cutting across the sternum with a Gigli saw. Both internal mammary arteries will have to be secured. The space selected will depend upon the site of entry. The standard approaches to the thoracic contents may also be used.

Incisions crossing flexion creases at the inguinal region, popliteal, and antecubital fossae should be interrupted by an S-shaped or transverse incision parallel to the flexion crease, in order to prevent scar contracture. This is more important in the Bantu who develop keloids and hypertrophic scars quite frequently.

Treatment of the Affected Artery

Before 1950, the treatment of arterial injuries was ligation.[4] The largest recorded series is that of De Bakey and Simeone[6] who analysed 2,471 injuries of the Second World War. The amputation rate was 50%. In 81 patients who were treated by lateral repair, or end-to-end anastomosis, the overall amputation rate was 35%.

The results of ligation of major arteries are shown in Table III. With the advent of antibiotics and abundant blood transfusions, surgical techniques developed by procedures such as repairing coarctations of the aorta and

TABLE III. RESULTS OF LIGATION OF MAJOR ARTERIES IN 2,471 CASES. (DE BAKEY AND SIMEONE[6])

	2,471 Cases	
Subclavian	28%	amputated
Axillary	43%	amputated
Brachial — Above profunda	55%	amputated
Brachial — Below profunda	25%	amputated
External iliac	46%	amputated
Common femoral	81%	amputated
Superficial femoral	54%	amputated
Popliteal	72%	amputated
Carotids	30%	(hemiplegia—death)

Fallot's tetralogy, and methods of storing arterial homografts. Arterial injuries were now approached with a view to repair.

In the Korean War and subsequently, these principles were put into practice on a large scale.

In Table IV a summary is given of the results of repairs of major arteries.

TABLE IV. RESULTS OF REPAIRS OF MAJOR ARTERIES (KOREAN WAR AND CIVILIAN INJURIES)

Hughes[3] 73 repairs, 8 amputations (1 upper limb)
Jahnke and Howard[8] 56 repairs, 6 amputations
Morris and De Bakey[1] (civilian injuries)
93 repairs, 7 amputations, 3 deaths
32 ligations, 5 amputations, 2 deaths
Seeley, Hughes, Cook and Elkin.[15] Aneurysms.
33 repairs, no amputations, 2·8% insufficiency
28 ligations, no amputations, 24% insufficiency

Acute arterial injuries after repair had an average overall amputation rate of about 10%.

Ligations had an amputation rate of about 20%.

The best results were obtained in fistulae and aneurysms where in large series amputations were a rarity.

The problem that any surgeon is faced with is: what arteries may be ligated, and what arteries have to be repaired. All arteries should be repaired, the only contra-indication being very poor condition of the patient.

Cranley has divided arteries into 3 groups[2] (Table V).

TABLE V. ARTERIES DIVIDED INTO THREE GROUPS AS REGARDS LIGATION (CRANLEY)

1. Tied with impunity
   External carotid
   Radial or ulnar
   One of arteries below popliteal trifurcation
2. Tied under certain circumstances
   Common carotid
   Vertebral
   Subclavian
   Axillary
   Brachial
   (*a*) Collaterals intact
   (*b*) Minimal soft tissue damage
   (*c*) Blood pressure maintained throughout
3. Repaired under all circumstances
   Aorta
   Innominate
   External iliac
   Femorals, popliteal

In the present series results should have been better than those obtained in battle injuries because of smaller calibre missiles, less frequent soft-tissue and bony injury, and earlier presentation.

Debridement of a lacerated artery, well clear of the affected part, is as important here as it is in the treatment of lacerated skin or muscle. Holman[7] advises resection of at least 1 cm. on either side of the laceration and has demonstrated degeneration including necrosis for some distance from the site of injury.

Mobilization of small collaterals in the vicinity is permissible to oppose the resected segments without tension, or only slight tension. Subintimal dissection should be transected well clear of its extremities. Anastomosis should be performed with atraumatic oiled silk 50, with at least one-third of the circumference being interrupted to allow for expansion or growth of the artery in young patients. Loose continuous sutures may be used. Adventitia should be stripped to allow suture of the media without interference with the anastomosis or inversion of the adventitia into the lumen. Potts or bulldog clamps should be avoided because of possible intimal damage they may produce. Proximal and distal control can be obtained by applying a firm catheter round the artery and securing it with artery forceps. Tapes may be used for the same purpose.

Spasm may be overcome by sympathetic block, local application of papaverine, 2·5%, or the injection of normal saline into the proximal segment under pressure to distend the spastic artery.[8] Deep fascia should be left unsutured at the end of the procedure, and where the affected limb is under tension fasciotomy should be performed. If in doubt this should be done. Heparin is generally not required systemically, but may be used locally in undiluted form to flush out the artery. It may be neutralized at the end of the operation by protamine sulphate 10 ml./5,000 u.heparin.

Autogenous vein grafts are adequate for any defect up to 4 - 5 inches. The vein should be reversed to offset the effects of valves that may be present. They are required in 10 - 15% of cases only.

Contusion with thrombosis should be resected because of the tendency for further thrombosis after thrombo-endarterectomy.[9]

Associated fractures should be stabilized by internal fixation by Küntscher-nailing or plating procedures. Limbs should not be encased in plaster of Paris; a slab will generally maintain adequate fixation. If plaster encasements are used they should be split down to the skin immediately after operation.

The results of arterial repair are shown in Table VI. Vein grafts were used in 8% of the present series and in 20% of repairs.

TABLE VI. RESULTS OF ARTERIAL REPAIR

A. Lateral suture or end-to-end anastomosis

	No.	*Functional*	*Failed but viable*	*Amputated*	*Died*
Upper limb ..	7	6	0	1	0
Lower limb ..	7	3	3	1	0
Aorta	2	1	—	—	1

B. Vein grafts

Carotid	1	—	—	—	1
Axillary	1	—	1	—	—
Femoral	2	1	—	1	0
Total	20	11	4	3	2

Absolute failures occurred in 5 cases (25%) and relative failures (viable extremities, failed repairs) in 4 cases (20%), the total number of occluded repairs being 9 (45%).

In arterial ligations the amputation rate was 14% with 33% remaining with ischaemic extremities, total complication rate being about 47% (Table VII). Ischaemic sequelae were much less where arterial repair was undertaken.

TABLE VII. RESULTS OF ARTERIAL LIGATION

	No.	*Normal*	*Deaths*	*Amputated (or hemiplegia)*	*Ischaemic*
Carotico-vertebral system	7	5	1	1	—
Upper limb ..	11	7	—	—	4
Lower limb ..	4	1	—	2	1
Total ..	22	13	1	3	5

The location and treatment of aneurysms and fistulae

are shown in Table VIII. The best results were obtained where the arterial injury had presented as an aneurysm or fistula, or where an acute injury was treated after an interim period after developing an aneurysm.

TABLE VIII. A. LOCATION AND TREATMENT OF ANEURYSMS AND FISTULAE

Site	*No.*	*Ligation*	*Repair*	*Results*
Carotid	2	1	1	Good
Vertebral	1	1	—	Good
Upper limb ..	7	4	3	Good
Lower limb ..	6	3	3	Good
Aorta	1	—	1	Good
Total	17	9	8	

B. ACUTE INJURIES TREATED CONSERVATIVELY

	No.	*Ligated*	*Repaired*
Carotid	2	1	1
Brachial	2	1	1
Femoral	3	3	0
Aorta	1	0	1
Total ..	8	5	3

Eight patients with acute injuries were treated conservatively because of small haematomas, pulses present distal to the affected artery, and small skin openings which did not allow of much external blood loss and in which soft-tissue injury was minimal.

The present consensus of opinion is that all acute arterial injuries should be explored initially,[10] but Hughes[6] is of the opinion that carotid artery injuries are best treated conservatively and allowed to develop a collateral circulation where the local condition allows.

In the present series those treated conservatively were operated on after an average of 35 days, the variation being 5 days to 5 months. All did well. Only 3 were repaired.

CAROTID ARTERIAL INJURIES

These present unique problems in that no matter what local procedure is adopted, whether ligation or repair, occlusion of the carotid circulation for as little as 3 minutes may result in irreversible cerebral softening causing death or hemiplegia.[11]

Harris and Udvarhelyi[12] and Brackett[13] in two separate series of a total of 131 cases of cerebral aneurysm, in which the carotid artery was ligated, had the following results: Death 10%, permanent hemiplegia 25%, and epilepsy or psychotic changes 15%. The incidence of these complications may be reduced to a bare minimum (approximately 5%) if several factors are considered:

1. *Cross-circulation test.* A contralateral carotid angiogram with ipsilateral compression (at operation if necessary) is performed to show if an adequate cross circulation (mainly *via* the anterior communicating artery) exists. If cross circulation is not present, ligation is fraught with danger.

2. *Drop in cerebral blood pressure* of as little as 10 mm. mercury may precipitate cerebral anoxia. This may occur with blood loss or during anaesthesia. Neurosurgeons often ligate the carotid artery under local anaesthesia for this reason (it also allows the early detection of hemiplegic signs, which may be reversed if the patency of the artery is immediately restored).[17] Important aids in the treatment of acute injuries as well as fistulae and aneurysms are hypothermia and an arterial bypass which should be of wide enough bore to allow good cerebral circulation.

VERTEBRAL ARTERIAL INJURIES

These may be of considerable importance because of their supply to the subtentorial structures.[17] Adequate collateral circulation is normally present provided both vertebrals and carotids are patent. In 5 - 10% of patients one vertebral artery is underdeveloped or absent. Ligation of the only normal vertebral artery in these circumstances may prove extremely hazardous. This anomaly may be detected only by angiography or direct exploration.[14]

Aneurysms may be treated by Hunterian ligation in the vertebral triangle or by trapping between ligatures or clips. Exposure of the vertebral artery in the neck may be obtained by nibbling the costo-transverse process.

In the present series 7 common carotid injuries occurred; 5 were operated on in the acute phase and 2 formed aneurysms or fistulae. Of the 5 operated on initially, 1 negative exploration resulted in hemiplegia which showed occlusion of the middle cerebral artery (a result of possible embolus from contused artery), and 2 were ligated without complications. One patient had a ligation and died of cerebral softening 12 hours later, and another with a saphenous vein graft died of the same cause 12 hours later, although the graft was patent at the end of the operation. The cerebral circulation had been occluded during the process of repair.

The 2 patients who developed aneurysm and fistula respectively were investigated by cross-circulation tests which were normal, and operated on under hypothermia. In the patient with a fistula the artery was repaired and the jugular vein ligated, and in the other patient the aneurysm was obliterated. Both patients made uneventful recoveries.

Two vertebral artery injuries were successfully treated by ligation in the vertebral triangle. No cross-circulation tests were done.

INCIDENCE OF SEPSIS

This is generally stated to be not very important. All the patients in this series received routine antibiotics (pencillin and streptomycin) and antitetanus serum.

In all cases where sepsis was a factor, thrombosis of the affected artery occurred. This may have developed without infection. Amputation was performed because of gas gangrene, not because of thrombosis (Table IX).

TABLE IX. INCIDENCE OF SEPSIS*

Type of infection	*No.*	*Amputation*	*Thrombosis*
A. Gas gangrene ..	3	3	3
B. Gross	1	1	1
C. Mild	2	—	2

* Total number of cases 6, i.e. 12%.

RÔLE OF ANGIOGRAPHY

The indications for angiography are summarized in Table X. Angiography plays little part in the acute injury before exploration in the detection of the site of the lesion. Direct angiography may be easily performed during operation.

TABLE X. INDICATIONS FOR ANGIOGRAPHY

1. Acute injuries
   After any form of repair to establish presence of occlusion (by clot) more distally (during operation). Performed twice.
2. Aneurysms and fistulae
   (*a*) Establish specific artery
   (*b*) Collateral supply
   (*c*) Diagnosis and venous filling
   Performed 16 times.
3. Long-term after-repair or graft
   Performed 8 times.
4. Cross-circulation test
   Performed twice.

Total no. 28.

It is not indicated to establish the presence of an adequate circulation in the first 24 hours after operation where pulses are absent. This may be surmised on clinical and oscillometric study alone.

Angiography should be performed as a long-term follow-up in arterial repairs to study the technique of anastomosis, length, and tortuosity of the graft and the occurrence of thrombosis.

SUMMARY

An analysis of 50 cases of major arterial injuries was made of patients presenting for treatment at Baragwanath Hospital over a 2½-year period.

Of these patients 3 died, 8 had amputations (one upper limb), 2 were left with permanent hemiplegias, 3 developed gas gangrene, and 5 were left with vascular insufficiency of the affected limb.

The complication rate for all the cases was approximately 40%. The type of treatment did not materially alter the result. The series is too small to come to any definite conclusions.

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THE TREATMENT OF PERFORATED PEPTIC ULCER

I. PENN M.B., B.CH. (RAND), F.R.C.S. (ENG.)

Senior Medical Officer in Surgery, Baragwanath Hospital and the University of the Witwatersrand. Formerly Surgical Registrar, St. James' Hospital, Balham, London

Perforation of a peptic ulcer is a common complication occurring in about 10% of all cases.[40] It threatens the life of the patient by producing first a chemical peritonitis, followed later by bacterial peritonitis, and early diagnosis and treatment are required if the patient is to survive.

In this paper I propose to review the literature on the subject, and to illustrate my remarks by referring to a series of 42 patients on whom I operated personally (Tables I and II).

Until recent years the treatment of perforated peptic ulcer consisted of simple closure of the perforation. However, with improved methods of fluid and electrolyte replacement, improvements in anaesthesia, and the introduction of the antibiotics, other forms of treatment have become increasingly popular.

At present there are 3 forms of treatment available: (1) conservative treatment, (2) simple closure of the perforation, and (3) radical surgery, including (*a*) immediate partial gastrectomy, and (*b*) immediate vagotomy combined with a gastric drainage operation.

Before discussing the various forms of treatment it is necessary to consider certain facts about perforated ulcers, since these have a bearing on prognosis and, therefore, on the choice of treatment.

1. *The Chronicity of the Ulcer*

It is important to distinguish between perforated acute and chronic ulcers, since there is a considerable difference in mortality and morbidity in the two conditions.[15]

They may be distinguished in two ways.

(*a*) *The history.* In acute ulcers there is generally no history of dyspepsia or only a very short one, whereas in chronic ulcers there is a history of chronic bouts of dyspepsia.

(*b*) *At operation.* In acute perforations the hole is small and punched out and the stomach and duodenal wall is mobile. In chronic ulcers the perforation is usually larger, and there is usually appreciable scarring and deformity of the adjacent stomach or duodenal wall. Thus, in some cases, despite a very short history of dyspepsia, it is obvious at laparotomy that the ulcer is a chronic one. This is illustrated by several cases in the present series. Case 14 gave no history of indigestion, but was found to have a chronic gastric ulcer at operation. Case 21 also had no indigestion, but operation showed a chronic duodenal ulcer, while case 36 gave a 6-weeks' history of indigestion and at operation a giant chronic gastric ulcer was found.

It has been shown that mortality occurs mainly in patients with chronic peptic ulcer. Thus Gilmour,[15] in a series of 119 cases treated by simple suture, and Taylor and Warren,[37] in a series of 47 cases treated conservatively, had no fatalities in patients with perforated acute ulcers, whereas in patients with chronic ulcers Gilmour[15] had a 15% mortality and Taylor and Warren[37] a 14% mortality. The reason for this is that in chronic ulcers factors such as chronic ill-health, electrolyte imbalance, and protein deficiency associated with pyloric stenosis, contribute to the fatal outcome.[33] In addition, it may be impossible to secure closure of the perforation owing to the rigidity of the affected area.[16]

In the present series there were 4 deaths, all of which occurred in patients with chronic ulcers.

2. *The Situation of the Ulcer*

Perforated gastric ulcers have a much worse prognosis than perforated duodenal ulcers.[3] Thus, in a series of 202 cases treated by simple closure, Desmond and Seargeant[12] found a 5·8% mortality in perforated duodenal ulcer, but a 21% mortality in perforated gastric ulcer. Gastric perforations are, on the whole,

TABLE I. PERFORATED PEPTIC ULCER TREATED BY SIMPLE CLOSURE

No.	*Age*	*Sex*	*Previous history*	*Type of ulcer*	*Duration of perforation*	*Remarks*
1	73	Male	?	Giant Chronic G.U.	6 hours	*Fatal haematemesis on 10th postoperative day. P.M.: Large benign G.U.
2	48	Male	?	Acute D.U.	3 hours	Uneventful recovery
3	57	Male	?	Chronic D.U.	5 hours	Had a previous perforation. Uneventful recovery
4	72	Female	?	Chronic D.U.	6 hours	Had haematemesis and melaena. 24 hours later had perforation. Uneventful recovery
5	63	Male	?	Chronic D.U.	4-6 hours	Uneventful recovery
6	62	Male	?	Chronic D.U.	4 hours	Uneventful recovery
7	65	Male	?	Chronic D.U.	8 hours	Uneventful recovery
8	46	Male	2 weeks	Acute D.U.	3 hours	Uneventful recovery
9	45	Male	1 year	Chronic G.U.	4 hours	Uneventful recovery
10	27	Male	Several days	Acute G.U.	±6 hours	Uneventful recovery
11	83	Female	?	Acute G.U.	12 hours	Certified mental patient. Uneventful recovery
12	42	Male	10 years	Chronic D.U.	3-4 hours	Patient under treatment for active pulmonary tuberculosis. Therefore treated by simple closure
13	10	Male	Several weeks	Acute D.U.	5 hours	Uneventful recovery. Still had indigestion one year later
14	72	Female	Nil	Chronic G.U.	8 hours	*Admitted with melaena. 10 days later perforation occurred while in hospital. There was a ½-inch perforation in the pyloric region. Died 36 hours after operation
15	18	Female	3 months	Acute D.U.	4 hours	Uneventful recovery
16	26	Male	6 years	Chronic D.U.	3 hours	Chronic alcoholic. Uneventful recovery
17	68	Male	Nil	Acute D.U.	18 hours	Uneventful recovery
18	63	Male	Nil	Acute G.U.	6 hours	Uneventful recovery
19	65	Male	3 months	Acute G.U.	10 hours	Mental patient. Had a previous gastrostomy for oesophageal stricture. Ulcer about one inch away from site of gastrostomy. Uneventful recovery
20	28	Male	9 months	Acute D.U.	4 hours	Uneventful recovery
21	56	Male	Nil	Chronic D.U.	5 days	*5 days abdominal pain, vomiting coffee grounds, melaena. Case of simultaneous bleed and perforation. Also chronic bronchitic. Gravely ill pre-operatively. Marked peritoneal soiling with a ¼-inch perforation. Died 48 hours after operation
22	27	Male	1 week	Acute D.U.	4 hours	Uneventful recovery
23	49	Male	Nil	Acute D.U.	4 hours	Previous history of bilateral Smithwick operation, nephrectomy, and coronary thrombosis. Uneventful recovery
24	48	Male	Nil	Acute D.U.	8 hours	Also found to have a carcinoma of the rectum. Abdomino-perineal resection 3 weeks later. Uneventful recovery
25	47	Male	10 days	Acute D.U.	5 hours	Uneventful recovery
26	49	Male	6 days	Acute D.U.	7 hours	Uneventful recovery
27	23	Female	2 weeks	Acute D.U.	3 hours	Uneventful recovery
28	48	Male	3 months	Acute D.U.	4 hours	Uneventful recovery

D.U.=Duodenal ulcer. G.U.=Gastric ulcer. *=Death. P.M.=Postmortem examination.

TABLE II. PERFORATED PEPTIC ULCER TREATED BY IMMEDIATE GASTRECTOMY

No.	*Age*	*Sex*	*Previous history*	*Type of ulcer*	*Duration of perforation*	*Remarks*
29	51	Male	4 years	Chronic D.U.	3 hours	Uneventful recovery
30	57	Male	9 years	Chronic D.U.	6 hours	Uneventful recovery
31	65	Male	30 years	Chronic D.U. with stenosis	1 hour	Uneventful recovery
32	46	Female	10 years	Chronic D.U.	10 hours	Uneventful recovery
33	64	Female	6 years	Chronic D.U.	8 hours	*Severe haematemesis and melaena 1 year previously. Operation advised but delayed in order that a radical mastectomy could be performed for carcinoma of the breast. Perforation treated by Moynihan antecolic partial gastrectomy. On 6th postoperative day developed severe abdominal pain. Laparotomy revealed strangulated loop of bowel behind anastomosis. Died 12 hours later
34	39	Male	10 years	Chronic D.U.	2 hours	Uneventful recovery
35	63	Male	20 years	Chronic D.U.	6 hours	Perforated anterior D.U., penetrating posterior D.U. and marked prestenotic diverticula. Small prepyloric ulcer and large hiatus hernia also present. Chronic bronchitic and alcoholic. Uneventful recovery
36	45	Female	6 weeks	Chronic G.U.	10 hours	Very pale. Pre-operative haemoglobin = 39%. Giant chronic G.U. with hour-glass deformity. Histology showed benign G.U. Uneventful recovery
37	55	Male	4 years	Chronic D.U.	6-7 hours	Uneventful recovery
38	76	Female	6 months	2 Chronic G.U.'s anterior and posterior	24 hours	Little peritoneal contamination. Uneventful recovery
39	79	Male	1 year	Chronic D.U.	12 hours	Uneventful recovery
40	79	Female	2 years	Chronic G.U.	8 hours	Uneventful recovery
41	39	Male	2 months	Chronic G.U.	21 hours	Marked peritoneal contamination. Postoperatively left subphrenic abscess drained. Thereafter uneventful recovery
42	44	Male	3 months	Perforated gastric carcinoma	48 hours	Gross peritoneal contamination. Histology: anaplastic carcinoma of stomach. Uneventful recovery

D.U. = Duodenal ulcer. G.U. = Gastric ulcer *=Death.

larger than duodenal perforations,[1] and in duodenal ulcers there is a much greater tendency for the perforation to become either partially or completely sealed off by a neighbouring viscus or omentum.[33] Thus, peritoneal soiling is often minimal in amount in perforated duodenal ulcers. In addition, gastric ulcers occur in patients in the older age group and in whom mortality is higher.

3. *The Question of Malignancy*

Quite a high proportion of carcinomas of the stomach appear in the guise of simple gastric perforations.[30] Thus, Doll[13] found 17 patients with perforated carcinomas of the stomach at the Central Middlesex Hospital in the years 1938–1948. Kennedy[21] quotes a series of 111 cases of perforation from the London Hospital, which includes 7 cases of perforated gastric carcinoma. Louw[22] states that 12% of gastric perforations are malignant. Taylor[38], in a series of 256 cases of perforation, found that the incidence of cancer was 2·3% of all perforations and 21% of all the perforations occurring in the stomach.

In the present series of 42 cases, 1 of the perforations was malignant. Often the growth is small and well localized without evidence of glandular or hepatic involvement, and is liable to be mistaken for a benign gastric ulcer.

TREATMENT

1. Conservative Treatment[4]

Rationale

The rationale for this form of therapy is that at operation one often finds that the perforation has already been sealed off by an omental plug or by adherence to an adjacent viscus. The aim of treatment is to preserve this seal and thus prevent further contamination of the peritoneal cavity; any bacterial invasion that has already taken place will be adequately dealt with by the peritoneum.

Method

By far the most important factor in conservative treatment is effective and constant gastric suction. This removes swallowed air and most of the gastric secretions, and also reduces the amount of duodenal secretion by removing the stimulus of gastric contents entering the duodenum. The seal of the perforation is left undisturbed and is allowed to become firm. This most important aspect of treatment requires constant supervision by the surgeon and nursing staff responsible.

The rest of the treatment consists of fluid and electrolyte re-

placement, administration of antibiotics, and suitable sedation.

The amount of gas under the diaphragm, on a plain X-ray film of the abdomen, is noted, and another X-ray is taken 12 hours later. If the second picture reveals an increase in the amount of gas, operation is considered.

The above treatment is continued until the patient has a soft, flat abdomen, normal bowel sounds are audible, and the patient has passed flatus. Gastric suction and intravenous fluid therapy are then discontinued and oral feeding commenced.

Surgery is only necessary to deal with a residual abscess (usually subphrenic), should this occur.

Advantages

1. *Fewer complications.* Wound infections, disruptions, and incisional hernias are eliminated, as are anaesthetic complications. There is no risk of postoperative adhesions, and pulmonary complications are markedly decreased. Intraperitoneal abscesses are no more frequent than in surgically treated cases, except for subphrenic abscess, which may have a higher incidence.

2. *Less stress.* The patient is subjected to less stress than with operative treatment. The possibility of acute coronary occlusion, or a similar disaster, is diminished.

3. Should surgery become necessary at a later date because of recurrent ulcer symptoms, the operative area is surprisingly free from adhesions and distortion. This makes the definitive operation much easier and, therefore, much safer. In addition, the patient can be operated upon when he is in the optimum condition.

4. An occasional complication of simple closure of a perforation is pyloric obstruction. This complication is almost non-existent with conservative treatment.

Disadvantages

1. *Uncertainty or error in diagnosis.* Errors in diagnosis may have serious consequences. Perforation of another viscus may mimic perforated peptic ulcer clinically, and there are cases of appendicitis and intestinal strangulation which were treated conservatively in the belief thay they were perforated peptic ulcers.

2. There is the possibility of inadvertently inserting the nasogastric tube through the perforation, and thus keeping it open. However, if the gastric aspirations are carefully recorded, this complication would be suspected by the limited amount of fluid being withdrawn. (Usually 1-2 litres of fluid a day are aspirated under these conditions.) In addition, the patient's symptoms would persist and the amount of gas under the diaphragm would increase. This objection is, therefore, more theoretical than real.

3. The fact that with non-operative treatment the site of the perforation is unknown, is another important criticism. Perforated gastric ulcers carry a higher mortality than perforated duodenal ulcers when operative treatment is employed, and this is equally true with conservative treatment. Beattie,[2] in a series of 40 patients treated conservatively, had 1 death out of 32 patients with duodenal ulcer, and 5 deaths out of 8 patients with gastric ulcer, one of the gastric ulcers being a perforated carcinoma.

4. Another objection is that there is a possibility of a perforated gastric carcinoma being treated conservatively. As the site of the perforation is unknown it is possible that an early carcinoma of the stomach (for example, malignant change in one part of a benign gastric ulcer) will be missed. Thus the chance of cure by performing an early gastrectomy will be lost.

5. The treatment will not affect the natural history of peptic ulcer, and many patients will subsequently develop recurrent symptoms which will require surgery. Taylor[38] found that after non-surgical treatment of perforated acute ulcer there was hardly any recurrence of symptoms, but in chronic ulcers there is a recurrent ulcer rate of no less than 85%, a partial gastrectomy rate of 60%, and an ulcer mortality rate of 4%.

Results

Seeley and Campbell[32] treated 139 patients with a mortality rate of 5%. Heslop *et al.*[17] made a carefully controlled study comparing two series of cases, each consisting of 104 consecutive and unselected patients. The mortality rate in the operative group was 8·6% while in the non-operative group it was 7·7%.

The proponents of conservative treatment claim that this form of therapy has at least an equal mortality rate, a definitely lower incidence of complications, and a more rapid return to normal activity as compared with the patients treated by surgery. However, examination of the results of other reported series does not confirm this favourable outlook.

I have already quoted Beattie's[2] series of 40 cases in which he had a mortality of 15%. Taylor[38] treated a series of 256 cases conservatively with 2·5% mortality in 79 patients with perforated acute ulcers, but with a 15% mortality in 177 patients with perforated chronic ulcers. A 4% mortality was added for subsequent deaths from recurrence, which made the ultimate mortality in patients with perforated chronic ulcers 19%. Taylor, therefore, reserves conservative treatment for perforated acute ulcers or for situations where operative treatment is impossible.

Höyer,[18] in a series of 97 cases, had a 50·5% mortality while Desmond and Seargeant,[12] in 12 unselected patients treated conservatively, had a 41% mortality.

The conservative treatment of perforated peptic ulcer has not received wide acceptance, partly because it has not lowered the mortality of this condition, and partly because of the great demands on the nursing and medical attendants for prolonged and anxious observation. It is unsuitable for routine usage.[36]

Conservative treatment is, however, useful in the following types of cases: (1) Where the patient refuses operative treatment, (2) where the patient is unfit for anaesthesia and laparotomy, and (3) where, for geographical reasons, surgery is not possible — for example, perforation occurring in an isolated country area or in a ship far out at sea.

In the present series no patients were treated conservatively.

2. Simple Closure[5]

Rationale

It is, firstly, a life-saving procedure and, secondly, will permit permanent ulcer control in many patients.

1. *Life-saving procedure.* It offers a safe and easy method of preventing further spillage of the gastric or duodenal contents into the peritoneal cavity. That it is a safe procedure is shown by the results of McCaughan and Bowers[26] who had a mortality of 1·5% in a series of 262 unselected cases.

Another advantage is that it can be more safely performed by the less experienced than can gastrectomy, and it can be performed in institutions with limited facilities.

2. *Permanent ulcer control.* After simple closure 35-40% of patients with perforated peptic ulcers will remain free of ulcer symptoms and will not require any further surgical treatment.

Method

The perforation is closed by a series of sutures inserted in the long axis of the stomach or duodenum. Where the tissues are poor and closure difficult, the closure may be reinforced by bringing up omentum and tacking it down to the sutured area. If suture is impossible by virtue of the rigid unyielding margins of the perforation, it is simply plugged with omentum. However, this latter procedure, although perhaps immediately successful, is unsatisfactory since it leaves an area of defective duodenal wall. This, in the presence of hyperchlorhydria, will inevitably re-ulcerate and perhaps re-perforate.[20]

Advantages of Simple Closure over Primary Resection

1. *Psychological preparation of the patient.* In order that resection may get the best possible results, it is necessary to spend some time in preparing the patient psychologically for the operation and its after-effects. It is impossible to do this if the patient is virtually 'dragged off the street' for the resection. Simple closure delays the definitive procedure and permits the necessary psychological preparation if gastric resection is needed in the future.

2. *Joy of living with a normal stomach.* Simple closure also has the advantage that in many cases the patient may remain symptom-free for some years before gastrectomy becomes necessary. During this period the patient is able to enjoy all the benefits derived from having a normal stomach.

Disadvantages of Simple Closure

1. *Obstruction.* In duodenal ulcers simple closure is unwise where considerable cicatricial narrowing is present or in cases with large perforations, since it may produce complete stenosis occurring either immediately or at a later date. Pyloric obstruction occurred in 13% of 107 patients followed up by Scholnick and Hastings.[31]

Thus, if the patient's condition will permit, immediate resection is the preferable procedure in such cases.

2. *Haemorrhage.* It is claimed that this is uncommon in duodenal

ulcers, since most perforated ulcers occur anteriorly and laterally—areas which are not as vascular as the posterior duodenal region. However, it can occur in encircling duodenal ulcers or in patients in whom two ulcers co-exist — the anterior one perforating and the posterior one bleeding. Scholnick and Hastings,[31] in a series of 107 cases treated by simple closure, found that major haemorrhage occurred in 22% of patients while 2·3% of patients died of bleeding peptic ulcer.

The danger of haemorrhage is illustrated by case 1 of the present series. A male, aged 73 years, was admitted with a large perforated gastric ulcer, the perforation having been present for 6 hours. The perforation was too large to close with sutures and was, therefore, closed with an omental plug. Postoperative progress was satisfactory until the tenth postoperative day when he had a massive haematemesis and died. Necropsy examination confirmed the presence of a large benign gastric ulcer.

3. *Recurrent perforation.* Scholnick and Hastings,[31] in 107 patients treated by simple closure, found that 6% of patients had recurrent perforation, while Matheson,[25] in 115 patients, found that reperforation occurred in 11%. Case 3 in the present series was admitted with a reperforation of his ulcer, having had his first perforation treated by simple closure some time previously. I have also seen a patient admitted with a third perforation, the two previous perforations having been treated by simple closure.

4. *Delayed gastric resection is made slightly more difficult* than in the average patient with uncomplicated ulcer, by virtue of adhesions about the site of the perforation. However, this is not a great drawback.

5. In many instances *the lesion is juxta-pyloric*, and at the time of perforation it is difficult, if not impossible, to determine whether the lesion is pre- or post-pyloric. The possibility of carcinoma subsequently developing in patients with pre-pyloric perforations must not be overlooked.

6. *Later re-operation.* One of the most potent criticisms of simple closure is that many patients develop recurrence of their ulcer symptoms and in many a further operation is necessary. Turner[39] found that 85% of patients had a recurrence of symptoms after simple closure, while Henley[16] found that only 1 out of 5 patients remained symptom-free after simple closure. Re-operation, thus, cannot be avoided in a large percentage of cases, but it does have the advantage that it is performed when the patient is in a more favourable condition.

7. If a *perforated gastric carcinoma* is treated by simple closure, widespread peritoneal metastases are often found at the subsequent laparotomy, and even a palliative gastrectomy is then inadvisable.[12]

Reviewing the above criticisms one is led to the conclusion that simple closure is not satisfactory as a routine procedure.

Indications for Simple Closure

1. *Perforated acute ulcers.* As stated above, recurrence of symptoms is a complication mainly of perforated chronic ulcers and is uncommon after perforation of an acute ulcer. Thus simple closure is the operation of choice in treatment of perforated acute ulcers.

2. *Perforated chronic ulcers*, where the patient is unfit to undergo gastric resection.

3. *Perforated stomal ulcers.* In the majority of patients simple closure is the treatment of choice, since it gives satisfactory results and also because primary gastric resection in these patients may be fraught with difficulties. Desmond and Seargeant[12] had good results in 6 out of 7 patients with perforated gastrojejunal ulcers treated by simple closure.

However, primary gastrectomy may prove necessary where the stomal ulcer is very large, or where the perforation is difficult to repair without distortion of the anastamosis.

3. Radical Surgery: Primary Gastrectomy[35]

Rationale

Not only does this deal with the perforation, but it also removes the ulcer, thereby sparing the patient the inconvenience caused by recurrence of his symptoms, and avoiding a subsequent operation in a large proportion of cases.

Simple Closure Compared with Primary Gastrectomy

In assessing the value of any surgical procedure the two most important factors for consideration are the late results and operative mortality.

1. *Late results.* The major objection to simple closure is that it does not alter the natural history of the disease, irrespective of the view, that was prevalent at one time, that once an ulcer perforates the healing process has been initiated. Statistics show a high incidence of recurrence of symptoms.

Illingworth *et al.*[19] found that remission of ulcer symptoms after perforation was seldom of long duration. Within 5 years 70% relapsed and 50% developed severe symptoms. The prognosis was found to be worse in patients with a long antecedent history, while the incidence of major complications, particularly reperforation and haematemesis, was 20% within 5 years of perforation.

Stabins,[35] in 167 cases, had a 65·1% recurrence of symptoms and 36·5% of his patients required further surgery later.

Emmett and Owen[14] found that in 70-85% of their patients in whom simple closure was done, recurrence of symptoms occurred, and about 40% required further surgical treatment for the relief of their persistent symptoms.

Höyer[18] in 430 patients, had a 72% recurrence of symptoms and 44% had a partial gastrectomy later.

Turner[39] found that 85·3% of patients treated by simple closure developed recurrence of symptoms. He stressed the dangers of reperforation and massive haemorrhage that exist in the immediate postoperative period, and advised gastric resection in the immediate postoperative period in those patients in whom primary gastric resection is contraindicated.

Tanner[36] found that, in a late follow-up of the patients with ulcer perforation treated by simple closure, one quarter of the patients with acute ulcer perforations had severe relapses of symptoms, and three quarters remained well, but of 65 patients with chronic ulcer perforations all had relapsed, two thirds requiring further operative treatment.

Thus, one is led to the conclusion that, with the exception of perforated acute ulcers, the late results of simple closure are far from satisfactory.

On the other hand, the late results of immediate gastric resection are very satisfactory and are probably no different from those following elective gastric resections.

Thus De Bakey[11] found, in a 1-5 year follow up of 74 patients treated by immediate gastrectomy, that the results were good or excellent in 89%, while Martinis, Olson and Harkins[24] found that 83% of their patients treated by primary or early elective gastric resection were completely asymptomatic on follow-up. Reports from other institutions similarly indicate a high percentage of good long-term results.

2. *Operative mortality.* Critics of primary resection argue that the procedure is too formidable for an acutely ill patient. They feel that the first duty of the surgeon is to save life and that control of the ulcer is a secondary consideration. They feel that there is no reason why a surgeon should attempt to perform a more difficult and more hazardous operation when a simpler safer closure procedure will keep the patient alive for a resection at a more propitious time.

Critics of resection argue that the mortality following simple closure is significantly less than that following resection. However, a review of the literature does not substantiate this contention.

Henley[16] treated 22 patients with perforated peptic ulcer by resection, with 2 deaths. In the same period 36 patients were treated by simple closure, also with 2 deaths.

Brachman, Cooley and De Bakey[6] treated 44 cases by resection with a 2·3% mortality. Noordijk[27] compared two series, the patients being of the same age group and having the same interval between perforation and operation. In 1,269 patients simple closure was carried out and in 777 gastrectomy. He found that the mortality rate was not significantly different in the two groups.

In the present series of 42 cases, 28 were treated by simple closure with a 10·7% mortality, while 14 were treated by primary gastrectomy with a 7·1% mortality.

De Bakey,[10] in his last 100 patients treated by primary gastric resection, had a mortality of 1%.

Nuboer,[28] in 131 patients with primary resection, had a mortality of 3·8%. It might be argued that it is impossible to compare the mortality rates of simple closure with resection, because simple closure is a routine procedure, whereas in resection a certain amount of selection of cases takes place. On the other hand, these mortality figures do not include the additional deaths that occur in patients who have had a simple suture of the perforation and either die later of further complications, such as reperforation or haematemesis, or who die later after being subjected to gastric resection for recurrence of their symptoms.

On the whole, increasing experience in many clinics has demonstrated that primary gastric resection may be applied with equal, if not with less, risk than the procedure of simple closure.

As regards the argument that emergency resection for perforation is more difficult and hazardous than elective resection, this, also, is not true. As Lowdon[23] pointed out, the anterior duodenal perforation is usually not associated with the technical difficulties so common with posterior and penetrating ulcers.

3. *Another objection to resection is that gastrectomy is unnecessary in many cases.* It has been shown that some 30% of patients are symptom-free after simple closure and most of these belong to the group of perforated acute ulcers, in whom a good result can be expected in the majority of cases. If the policy were adopted of performing a primary gastrectomy in all cases, it would involve an unnecessary mutilation in this 30%. Obviously, therefore, cases must be selected. In patients with chronic ulcers the majority will require further surgery after simple closure and there is, therefore, a very good case for performing primary gastrectomy in these patients, and simple closure in patients with perforated acute ulcers.

Factors Influencing the Choice of Patients for Primary Gastrectomy

Before undertaking emergency gastrectomy certain factors have to be considered.

1. *The surgeon.* It is essential that the surgeon has adequate experience of performing gastrectomy and is working in an institution with adequate facilities for good operative and postoperative care of the patient. If emergency gastrectomy is undertaken by inadequately trained surgeons in ill-equipped institutions the mortality figures would rise to prohibitive levels and would bring the operation into disrepute.

2. *Age.* Mortality increases with age. Desmond and Seargeant[12] found no mortality under the age of 40 years, while above this age the mortality of perforation steadily rose to 80% in patients over 80 years of age. Berne and Mikkelsen[3] found that the mortality of perforation was 4% for patients under 50 years of age and about 16% for patients over 60 years. As a general rule, in the elderly the smallest possible operation, namely, simple closure, should be performed. However, age, *by itself*, need not be a determining factor. Other factors besides age have to be taken into account and each case has to be treated on its own merits. Thus, in the present series, 3 patients were aged 76,79 and 79 years, but since all were in good general condition, primary gastrectomy was performed and all made a good recovery.

3. *Duration of perforation.* The ideal time to perform resection is within the first 12 hours, since it has been shown that the mortality increases considerably with perforations of longer duration. As a general rule, we are reluctant to perform gastrectomy in cases where the perforation has been present for more than 12 hours. However, duration need not, *in itself*, be a determining factor. It often happens, especially in duodenal perforations, that partial or complete sealing off of the area by a neighbouring viscus has occurred. In these cases peritoneal contamination is often surprisingly small, even 12 hours after perforation, and what infection is present can be controlled by antibiotics and by fluid and electrolyte replacement. Thus de Bakey[9] found that 75% of gastrectomies were performed in the first 12 hours after perforation and 10% as late as 24 hours after perforation.

As a general rule, a gross degree of peritoneal soiling is a contra-indication to resection, but this need not always be a deterrent. For example, Tanner[30] reported successful cases of primary resections in late perforations with frank pus in the peritoneal cavity. Rowlands and King[20] had a similar experience in 2 patients.

In case 41 in this series the patient had a perforated chronic gastric ulcer for 21 hours with gross peritoneal soiling and frank pus. A primary resection was performed. Postoperatively a left subphrenic abscess required drainage, otherwise recovery was uneventful.

In case 42 in this series the patient had a perforated gastric carcinoma of 48 hours' duration. There was a considerable amount of frank pus in the peritoneal cavity. Primary resection was followed by an uneventful convalescence apart from mild wound sepsis.

4. *The size of the ulcer* plays no part in the final decision unless it is an acute ulcer, in which case simple closure is all that is necessary. The great majority of perforated duodenal ulcers are situated on the free border of the duodenum and constitute no unusual technical difficulty. It is usually easy to free the duodenum, pylorus, and stomach from the inflamed and oedematous surrounding tissues. Closure of the duodenal stump in perforated duodenal ulcers is usually easy, though it is necessary in most cases to perform an open division and closure of the duodenum, since the oedema of its walls and the size of the perforation prevent the application of a clamp either across the ulcer or distal to it. Emmett and Owen[14] confirm that the technical difficulties of gastrectomy performed as an emergency for perforation are no greater than that performed as an elective procedure. A similar view is expressed by Louw.[22]

It is important to stress these points since the idea seems to be current that immediate gastrectomy in these cases is a hazardous procedure with a stormy postoperative course to be expected.

Selection of Cases for Gastrectomy

In *selecting cases for gastrectomy* the criteria of Desmond and Seargeant[12] are useful. Gastrectomy is carried out in the following cases:

1. *Perforated chronic gastric ulcer.* (Because of the high mortality of simple closure and because of the 12% risk of the ulcer being malignant.)

2. *Perforated gastric carcinoma.* It is usually assumed that when a gastric carcinoma perforates, it liberates viable neoplastic cells into the peritoneal cavity, which then form widespread peritoneal implants. However, Doll[13] has shown that this does not necessarily always occur, and this point is given further emphasis by Emmett and Owen's[14] patient with perforated gastric carcinoma who remained well for 6 years after primary resection.

3. *Perforated chronic duodenal ulcer* (for the reasons already given). There are several subgroups under this heading:

(*a*) In patients over 40 years of age with a history of dyspepsia of 6 months or more.

(*b*) In patients under the age of 40 years with a history of dyspepsia of at least 12 months.

(*c*) In patients with perforated duodenal ulcer with a short history, when the ulcer at operation is obviously a chronic one.

4. *Perforation combined with haemorrhage.* This double complication is said to occur in between 1-13% of patients.[34] The combination is very dangerous, the co-existence of haemorrhage increasing the mortality.[20] This combination was present in cases 4, 14, and 21 of the present series. All were treated by simple closure, 2 patients (cases 14 and 21) died.

4. Radical Surgery: Vagotomy and Gastric Drainage.

Rationale

Vagotomy plus a gastric drainage operation has become increasingly popular in the definitive treatment of chronic duodenal ulcer. Davies[8] has reported favourable results while Burge[7] considers it the operation of choice in the treatment of chronic duodenal ulcer. In recent years vagotomy has been introduced in the treatment of perforated duodenal ulcers. Not only does this form of therapy deal with the perforation, but it also provides a permanent cure of the ulcer.

The operation may take one of two forms.

(*a*) At operation the perforation is carefully inspected and, if the duodenum appears suitable for pyloroplasty, a Heineke—Mikulicz pyloroplasty followed by subdiaphragmatic vagotomy is performed.[29]

(*b*) Alternatively, the perforation is treated by simple closure. Then immediate subdiaphragmatic vagotomy and a gastro-enterostomy is performed.[7]

Advantages

1. It saves the patient a subsequent operation for the control of recurrent ulcer symptoms and it provides permanent control of the ulcer in the vast majority of cases.

2. It is an operation of considerably less magnitude than partial gastrectomy.

3. The stomach is not removed and postoperatively the patient's nutrition is better than in patients who have had a gastrectomy.

Disadvantages

1. In performing vagotomy the posterior mediastinum is opened, and infected material from the peritoneal cavity may be introduced into this space giving rise to mediastinitis.

2. Where pyloroplasty is performed to provide gastric drainage, it is possible that leakage may occur owing to the duodenal wall being fibrosed and rigid due to the presence of a chronic duodenal ulcer.

3. The incidence of recurrent ulceration is higher than that following partial gastrectomy.[6]

Results

Pierandozzi, Hinshaw and Rogers[29] treated 36 patients with an average age of 38 years and an average duration of perforation of 9 hours. There were no deaths and no evidence of postoperative duodenal leaks or fistulae. There were no symptoms or signs of mediastinitis, while a residual abscess developed in 1 patient. The procedure was not used in patients over 55 years of age, in late perforations, if there was excessive peritoneal spill, in patients who remained in shock, or in those with severe systemic diseases or alcoholism.

Vagotomy and gastric drainage was not used in treating any cases in the present series.

As yet no one has had sufficient experience with vagotomy and a gastric drainage operation in the treatment of perforated chronic duodenal ulcers to make any dogmatic statements, but further experience may show that it is a very satisfactory method of treating such cases.

SUMMARY

1. The mortality of perforated peptic ulcer occurs mainly in chronic ulcers.

2. Perforated gastric ulcers have a higher mortality than perforated duodenal ulcers.

3. Some 12% of perforated gastric ulcers are malignant.

4. Conservative treatment is indicated where: (*a*) the patient refuses surgery, (*b*) surgery is impossible for geographical reasons, and (*c*) the patient is unfit for surgery.

5. Simple closure is indicated in: (*a*) perforated acute ulcers, (*b*) perforated chronic ulcers where the patient is unfit for gastrectomy, and (*c*) most perforated stomal ulcers.

6. Immediate gastrectomy is indicated in: (*a*) perforated chronic gastric ulcer, (*b*) perforated gastric carcinoma, (*c*) perforated chronic duodenal ulcer, and (*d*) where bleeding is present in association with perforation.

7. The place of vagotomy and a gastric drainage operation in the treatment of perforated duodenal ulcer has not, as yet, been settled.

8. A personal series of 42 cases is presented. Of these patients, 28 were treated by simple closure with a 10·7% mortality and 14 by immediate gastrectomy with a 7·1% mortality.

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38. Taylor, H. (1957): Gastroenterology, **33**, 353.
39. Turner, F. P. (1951): Surg. Gynec. Obstet., **92**, 281.
40. Werbel, E. W., Kozoll, D. D. and Meyer, K. A. (1947): Surg. Clin, N. Amer., **27**, 93.

WORLD LIST OF FUTURE INTERNATIONAL MEETINGS

ADDITIONS AND ALTERATIONS NOTIFIED DURING MAY 1961

International Congress of Practical Medicine, Merano, Italy, 21 August-2 September 1961. Bundesärtzekammer, 1 Hädenkampfstrasse, Cologne, Germany.

World Federation of Neurology, Rome, September 1961. Dr. A. Lowenthal, Institute Bunge, Berchem-Antwerp, Belgium.

5th Pan American Congress of Endocrinology, Lima, 15-21 October 1961. Dr. M. San Martin, Secretary-General, Av. Central 325, San Isidoro, Lima, Peru.

QUESTIONS ANSWERED : VRAE BEANTWOORD

DISINFECTION OF OPERATING THEATRES

Q. — *What does the spraying of an operating theatre with formalin achieve? Are there any better agents than formalin? If spraying achieves a useful purpose, what are the indications for spraying?*

A.—These questions are difficult to answer without further information as to what is implied by 'spraying'.

As an aerial disinfectant and as a spray for walls and furniture, formalin is not suitable because of its extremely irritant properties and lack of penetration.

More effective and more practical spraying is obtained by using one of the newer quaternary ammonium compounds with residual disinfectant properties.

Spraying is useful in reducing bacterial contamination. It is common practice to employ spraying at the end of each operating session and after septic cases. Many institutions have separate theatre facilities for septic cases where spraying is done after each operation.

PASSING EVENTS : IN DIE VERBYGAAN

The Japanese Society of Tropical Medicine, 3rd Meeting, will be held in Tokyo from 10 to 13 September 1961. The programme of this congress will include panel discussions of tropical hygiene, control of malaria and filariasis, and venomous snakes and venom. The American Express Company, 34 Burg Street, Cape Town, have been appointed as official agents covering this congress, and all enquiries should be addressed to them.

* * *

The Southern African Cardiac Society. At a meeting of the Southern African Cardiac Society (Cape Town), held at Groote Schuur Hospital on Thursday 25 May 1961, the following was the programme:

1. Dr. Maurice Nellen described the technique of transatrial septal puncture, as described by Ross and Morrow, of the National Heart Institute, Bethesda, Maryland. Gratitude was expressed to Dr. Peter Nixon and Mr. Geofrey Wooler of the Thoracic and Cardiac Departments, Leeds General Infirmary, for their instruction.

In addition, Dr. Nellen gave a talk on the papers presented at the meeting of the British Cardiac Society, which he attended recently in Bristol.

2. Dr. Walter Beck presented a paper on dextro-cardia, dextro-rotation, and dextro-version.

3. Dr. V. Schrire described a case in an infant on whom Dr. Chris Barnard successfully operated for a coronary artery right ventricular aneurysm.

4. Dr. Walter Beck described and discussed a case of supravalvular aortic stenosis.

5. *Announcements*. The Chairman, Dr. M. Nellen, informed the meeting that Dr. John Goodwin, Secretary/Treasurer of the British Cardiac Society, informed him that the British cardiologists will be chartering one or two private airliners from London to Mexico in October 1962, and that South African cardiologists could be included in this concession.

Dr. Len Braudo, National Secretary of the Southern African Cardiac Society, has been informed of this offer.

* * *

Mr. Irmin Henkel, orthopaedic surgeon, of Pretoria, is leaving on a short visit to England and the Continent on 24 June 1961. On his return, he will continue to practice at a new address, 330 Robert Kock Medical Building, Pretorius Street, Pretoria, as from 1 August 1961. His telephone numbers will remain unchanged.

Dr. Irmin Henkel, ortopediese chirurg, van Pretoria, vertrek op 24 Junie 1961 op 'n kort besoek na Engeland en die Vasteland. Na sy terugkeer sal hy sy praktyk voortsit by 'n nuwe adres: 330 Robert Kock Mediese-gebou, Pretoriusstraat, Pretoria, vanaf 1 Augustus 1961. Die ou telefoonnommers sal onveranderd bly.

Edenvale Hospital, Johannesburg. The next clinical meeting will be held on Wednesday 21 June at 8.15 p.m. in the Board Room. The meeting will take the form of a symposium on alcoholism. Dr. I. Bersohn will speak on 'Alcoholism and liver tests'; and Dr. M. C. Frame will speak on 'Clinical management'. A film will be shown, followed by a discussion opened by Dr. Z. Wolf. All those interested are invited to attend this meeting.

* * *

South African Institute for Medical Research, Johannesburg, Staff Scientific Meeting. The next meeting will be held on Monday 26 June at 5.10 p.m. in the Institute Lecture Theatre. Dr. J. Metz will speak on 'Vitamin B^{12}, folic acid and megaloblastic anaemias'.

* * *

Lede word daaraan herinner dat hulle die Sekretaris van die Mediese Vereniging van Suid-Afrika, Posbus 643, Kaapstad, sowel as die Registrateur van die Suid-Afrikaanse Geneeskundige en Tandheelkundige Raad, Posbus 205, Pretoria, moet verwittig van enige adresverandering. Versuim hiervan beteken dat die *Tydskrif* nie afgelewer kan word nie. Dit het betrekking op lede wat oorsee gaan sowel as dié wat binne die Unie van adres verander.

* * *

Mr. Samuel Skapinker, specialist surgeon, of Johannesburg, has returned from a short visit to the USA, where he visited the Lahey Clinic, Boston; the Mayo Clinics, Rochester; and surgical centres in New York.

* * *

Mr. H. Perkin, specialist surgeon, of Cape Town, has changed his address to 602 Medical Centre, Heerengracht, Cape Town. The telephone numbers will remain unchanged.

Dr. H. Perkin, chirurg, van Kaapstad, het sy adres verander tot Mediese-sentrum 602, Heerengracht, Kaapstad. Die ou telefoonnommers bly onveranderd.

* * *

Dr. C. J. B. Muller, radioloog, en Hoof van die Departement van Radiologie, Universiteit van Stellenbosch, het teruggekeer van 'n besoek aan die Verenigde Koninkryk en die Vasteland waar hy in verskillende sentra gedurende die afgelope 6 maande kobalt- en ander hoë energie-bestraling bestudeer het.

* * *

Vereniging van Chirurge van Suid-Afrika (M.V.S.A.), Pretoria Sub-group. Die volgende vergadering van hierdie sub-groep sal op Vrydag 23 Junie om 5 nm. in die Kliniese-gebou, Pretoria, plaasvind. Dr. F. Ziady sal die vergadering toespreek oor ‚Haemotologiese siektes van chirurgiese belang'.

* * *

Dr. Lilian Raftery, M.R.C.O.G., M.M.S.A., M.R.C.S., L.R.C.P., was admitted, in absentia, to the Fellowship of the Royal College of Obstetricians and Gynaecologists at the meeting of the Council held in London on 27 May 1961.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

MICRIN

Johnson & Johnson (Pty.) Limited announce the introduction of Micrin, and supply the following information:

A new principle of oral antisepsis has emerged from a large and extensive laboratory screening programme by Johnson & Johnson in the USA. This programme was directed towards finding the most powerful, the safest and most long-acting combination of antimicrobial agents. Johnson & Johnson will soon be introducing Micrin to the Southern African market as a result of this research programme.

Their research staff discovered that 'dequalinium' [registered Trade Mark for Decamethylene-bis-(4-aminoquinaldinium acetate)], when used as an oral antiseptic gives a valuable benefit, *viz.* the *prolonged* suppression of odour-forming bacteria. In Micrin, 'dequalinium' is used as a complement to cetyl pyridinium chloride, which kills most of the common micro-organisms in the mouth within 30 seconds. Many of these organisms, however, survive because they are protected by the mucus and mucosal folds, etc., and unless these survivors are suppressed they can rapidly supplant the millions of organisms which have been killed by brief contact with the antiseptic. Dequalinium provides the necessary inhibitory action. It is bound rapidly by the mucosal lining of the mouth and, if gargled, by the mucosa of the pharynx, and is released slowly into the saliva to prevent the multiplication of the organisms for several hours after use.

Clinical testing of Micrin in individuals has provided evidence for:

(*a*) Safety in frequent and routine usage.

(*b*) Control of oral micro-organisms by rapid reduction in numbers and particularly in prolonged suppression of survivors. Effective control of orally caused bad breath is evidenced by a reduction in intensity of odour as well as a lasting suppression of odour.

(*c*) Since the micro-organisms are the agents forming the acid that is considered to be the major cause of tooth decay, a beneficial effect may be realized as a result of Micrin's inhibitory action.

(*d*) Symptomatic relief of minor sore throats caused by colds.

(*e*) A frequent beneficial effect on gingival colour, tenderness and tone.

As a result of test marketing, Johnson & Johnson have established an immediate consumer acceptance of Micrin. Users have expressed a definite liking for its fresh mint taste and have appreciated the fact that it does not taste 'like an antiseptic'. Micrin was released on the American market at the beginning of this year and will be available throughout Southern Africa during the later part of June this year.

Further information may be obtained from Johnson & Johnson (Pty.) Ltd., P.O. Box 727, East London.

BETNELAN

Glaxo-Allenburys (S.A.) (Pty.) Limited introduce an entirely new anti-inflammatory steroid and supply the following information:

Betnelan is 16-beta-methyl-9 alpha-fluoro-prednisolone, for which the British Pharmacopoeia Commission has approved the name betamethasone. Betnelan is about 8 times as potent as prednisolone, but this ratio varies, and in individual cases it may be no more than 5 times as potent or as much as 15 times as potent as prednisolone, although in most cases the ratio will be between 7 and 9 to 1.[1]

Apart from the enhancement of therapeutic potency, the principal object of chemical modification has been to reduce mineralocorticoid activity—sodium retention and potassium depletion. Betnelan represents the greatest achievement in this respect, being devoid of sodium-retaining activity, and in normal therapeutic use no significant potassium loss need be anticipated. There has been an indication that Betnelan may show less tendency to cause gastric intolerance,[1] but in this and other respects it must be accepted that no definite opinion can be formed until the substance has been in general use for an adequate period of time. Betnelan has no depressive effect on mental function, and patients receiving this corticoid have stated that they felt better, especially those transferred from triamcinolone.

Betnelan is capable of being used in a wide range of dosage according to the circumstances in which it is employed. Therapy is frequently initiated with 1 or 2 tablets 3 or 4 times daily, but if treatment is to be continued it is very desirable to determine the minimum acceptable dose. Betnelan is virtually non-toxic in the usual sense, in that large amounts can be taken as a single dose, or over a period of a few hours, without toxicity. Side-effects arise essentially from natural hormonal activity, develop gradually, and are therefore associated with long-term therapy. With modern dosage schemes such side-effects do not usually represent a serious impediment to treatment.

Short-term therapy can be used, for example, in:

Acute asthma and hay fever; severe eczema; urticaria and other allergic manifestations, including drug reactions; and shock, including toxaemic shock in overwhelming infections. The dosage commonly employed in these conditions is 2 tablets *t.i.d.* for 2 days, followed by 1 tablet *t.i.d.* for 2 days, and ½ tablet *t.i.d.* for 2 days.

In rheumatoid arthritis, the dosage could be 1 tablet 3 or 4 times a day for a week or two and thereafter the dosage might be reduced in successive stages until the minimum dosage capable of providing adequate effect is determined. Full use of aspirin and/or other analgesics should be made to reduce the dose of steroid needed.

Betnelan can be extremely useful in the nephrotic syndrome, also in congestive heart failure, since it is lacking in sodium-retaining activity or tendency to cause potassium loss. The dosage may be 1 or 2 tablets 3 or 4 times daily for 1 to 3 weeks, treatment then being tapered off gradually.

Betnelan is available in 0·5 mg. tablets in bottles of 30, 100 and 500. Prices are considerably lower than those of other branded steroids, the public prices for the packs mentioned above being respectively R2.10, R6.30, R28.35.

1. Glyn, J. H. and Fox, D. B. (1961): Brit. Med. J., **1**, 876.

TRYPTANOL

Merck Sharp & Dohme announce the discovery and development of Tryptanol, and supply the following information:

This drug represents a significant advance in the treatment of depression and especially depression accompanied by anxiety.

Tryptanol, amitriptyline hydrochloride, is chemically designated as 5-(3-dimethylaminopropylidene)-dibenzo[a,d]1,4-cycloheptadiene hydrochloride.

Its effect is particularly noticeable when anxiety and agitation have emerged as the dominant symptoms in a depressed patient.

It has been asserted that: 'Central-nervous-system stimulants and antidepressants, if given to anxious patients, will increase the anxiety . . . .'[1] Tryptanol, on the contrary, when used in this type of patient '. . . .acted both as a tranquillizer and as an antidepressant'.[2]

Also, unlike other potent psycho-pharmacological agents, Tryptanol achieves its wide range of therapeutic goals with a remarkable degree of safety. It has not been observed to produce parkinsonism, dystonia, agranulocytosis or jaundice. Moreover, none of the highly undesirable and sometimes bizarre side-effects seen with the phenothiazines, reserpine, barbiturates, and meprobamate may be expected to occur with Tryptanol, since it differs from these agents both chemically and pharmacodynamically. In comparison with imipramine, Tryptanol '. . . .appears to have a greater therapeutic activity', and since 'generally the side-effects are less severe with Tryptanol. . . .more patients can tolerate this drug'.[3]

The parenteral form of Tryptanol has been useful in the prompt control of anxiety, and in many instances has obviated or reduced the need for electroshock therapy.

Tryptanol is supplied in two forms: as tablets, each containing 10 or 25 mg. of amitriptyline hydrochloride; and as an injection for intramuscular or intravenous use. Each c.c. contains 10 mg. of amitriptyline hydrochloride.

Further information is available on request from Merck Sharp & Dohme, (Pty.) Ltd., P.O. Box 7748, Johannesburg.

1. Perloff, M. M., and Levick, L. J. (1960): Clinical Med., **7**, 2237.
2. Freed, H. (1960): Amer. J. Psychiat., **117**, 445.
3. Ayd, F. J., Jr. (1960): Psychosomatics, **1**, 320.

BOOKS RECEIVED : BOEKE ONTVANG

Children for the Childless. Ed. by M. Fishbein, M.D. Pp. xiii+210. R1.25 net. London: William Heinemann Medical Books. 1960.

Aids to Surgical Anatomy. 5th edition. By D. B. Moffat, M.B., B.S., F.R.C.S. Pp. 220. Illustrated. R1.20. Postage: R0.10. London: Baillière, Tindall and Cox. 1960.

CORRESPONDENCE : BRIEWERUBRIEK

WIDENING THE SCOPE OF MEDICAL AID SOCIETIES

To the Editor: The general practitioners will be receiving from approved medical aid societies and insurance companies an extra 25c for consultations and domiciliary visits, provided this tariff is preferential.

This is especially welcome at the present stage of financial stringency. Credit for this must be given to Federal Council and its important Subcommittee, namely, the Central Committee for Contract Practice (CCCP).

As a new member of Federal Council and CCCP, I was very impressed by the efforts of many specialists on this Committee who made the increase in general practitioners' fees their sole and main objective. So were the representatives of the medical aid societies. I was also informed by representatives of medical aid societies that this increase would have geen granted some time ago if Federal Council had taken a more realistic attitude in the past on the upper ceiling limit for medical aid societies and the incorporation of new societies.

I would plead with certain agitators in our profession, who seem intent on splitting the Association and attempting to undermine most of the decisions of Federal Council and its most important committee, the Executive Committee, to exercise more restraint and judgment in their criticisms, which are usually unfounded, misleading, or inspired by personal motives.

Our Federal Councillors are the properly elected representatives of the whole profession. We must recognize the authority that the members delegate to Federal Councillors and we must have confidence in their judgment and in the motives that inspire them when negotiating with medical aid societies and insurance companies. The decisions are not taken without careful deliberation of all the facts and circumstances that are pertinent to the many issues that are involved.

There is an unfortunate trend in some Branches to undermine and to try to rescind the recent agreements, particularly that made with SANSOM: I have no hesitation in saying that, unless the profession creates the opportunity for members of the public to belong to a sick fund, a State type of medical service will be enforced on the profession by public opinion and by the Government.

It is for this reason as well as for the welfare of the practice of medicine that I, for one, shall not cease to press for a widening of the scope of medical aid societies and the medical insurance schemes.

Marquard de Villiers

618 Medical Centre
Pretorius Street
Pretoria
1 June 1961

ENDOTRACHEAL ANAESTHESIA

To the Editor: I cannot allow Dr. Jones' article[1] in the *Journal* of 27 May to go unchallenged. How he arrives at his conclusions is beyond my comprehension.

If endotracheal intubation is to have ill-effects, these effects will take place at the time of intubation, and *not* 20-50 minutes later. So cases A., E. and F. in his series are completely out of context. As for cases B. and C. no autopsy report was available, so he, with us, can only speculate on the cause of cardiac arrest. In case D. it was obviously the concomitant anoxia, and *not* the endotracheal intubation which caused the after-effects.

If mechanical difficulties are present, anaesthetization to the third plane of the third stage of ether anaesthesia will *not* afford any advantages over the thiopentone-relaxant technique. Also, if vagal hypersensitivity is present, anaesthesia in this plane will afford no greater protection than the 'crash' technique. This is illustrated by the following case:

A child, aged 15 months, was admitted for repair of a cleft palate. Examination revealed no other abnormalities. Atropine, gr. 1/200, was given 45 minutes before the operation, and anaesthesia was induced and maintained with nitrous oxide, oxygen and ether until well into the third plane. An endotracheal tube was passed nasally under vision without any trouble, and she suddenly went very pale. Not realizing the cause, I switched off the nitrous oxide and ether and gave her oxygen under pressure, using the open lip of the T-Piece, with the tube in position.

Colour gradually improved and she was eventually sent back to the ward for further investigation. A blood count showed a very slight anaemia, which was corrected, and she came for operation 6 weeks later.

On this occasion, she was given scopolamine, gr. 1/300, instead of atropine, and rectal pentothal. She was anaesthetized well into the third plane with ether and an endotracheal tube passed, with the same sequence of events. As we were in doubt about the cause, it was again decided to abandon operation for 2 days, and to call in a paediatrician to do continuous ECGs.

She was duly anaesthetized on this occasion and during induction and maintenance there were no ECG changes. The tube was passed with no trouble, and again the pallor ensued with marked slowing and irregularity of the ECG, showing vagal hyperactivity. The tube was removed, the vocal cords, larynx, and trachea well sprayed with 5% cocaine, the tube reinserted, and the anaesthesia and operation from this point proceeded to a smooth and satisfactory conclusion.

If Dr. Jones objects to the thiopentone-relaxant induction because of vagal over-activity, the solution lies in his own hands. He has merely to spray with a surface anaesthetic and lubricate the tube with an anaesthetic ointment. It seems pointless to condemn a method which has so many advantages by quoting irrelevant cases in his article.

H. Bentel

12 Jubilee Road
Parktown
Johannesburg
6 June 1961

1. Jones, C. S. (1961): S. Afr. Med. J., 35, 421.

ENDOTRACHEAL ANAESTHESIA

To the Editor: It came as something of a shock to read a main article in the *Journal* by Dr. C. S. Jones.[1] He has made an attack on standard modern anaesthesia. If one were to be influenced by this article it would set the clock back at least 15 years.

Now, the gist of the article by Dr. Jones is that it is dangerous to intubate with the aid of a muscle relaxant, and that it would be safer to anaesthetize the patient to the second plane of surgical anaesthesia with ether before intubation. This is robbing Peter to pay Paul, because ether is a well-known peripheral 'curarimimetic'.[2] This sort of advice further neglects the nausea and vomiting, explosion hazard, and the delayed arousal associated with ether.

In the practice of anaesthesia the greatest danger is inadequate ventilation leading to hypoxia or hypercarbia, or both. Thus, the significant factors responsible for the appearance of ventricular arrhythmias with, or following, endotracheal intubation were found to be increased carbon-dioxide tension, lowered blood arterial oxygen, or both.[3] This is the rule during second plane surgical anaesthesia or at any rate sufficiently deep ether-anaesthesia for intubation, and it has long since been established that a specific muscle relaxant is far safer than ether or any potent general anaesthetic for intubation in a patient merely rendered unconscious.[4,5] How can a method which avoids the toxicity of deep anaesthesia, which ensures a free airway and which allows assisted or controlled respiration, add to the danger of an anaesthetic?

While reading the 6 case-reports in Dr. Jones' article one becomes less convinced than ever that laryngeal intubation had anything to do with the cardiac troubles. One cannot assume that the intubation was carried out atraumatically and with the requisite ability to do so, because this is doubtful in at least case 'D'. An oral route had failed, and a blind nasal approach substituted. Then cyanosis of

considerable duration followed! The patient was obviously subjected to a great deal of anoxia and hypercarbia, and one may well wonder what part of the anatomy it was that was 'blindly intubated'. Again, in case 'E', the apprehensive patient was given only atropine as premidication, and then anaesthetized with 'pentothal', nitrous oxide, and, mainly, trichlorethylene. Hyperpnoea developed and after 40 minutes the pulse was absent. Why not blame the 'trilene'? Under the same circumstances 'trilene' has often been responsible for ventricular fibrillation and death. Some years ago 6 deaths were reported in the west of Scotland from 'trilene'.[6] In case 'F' a brain operation was performed for a lesion in the vicinity of the fourth ventricle. Because the patient became a mental cripple the intubation technique is distrusted. What about the surgery? What are the grounds here for blaming the anaesthetist? In fact, why blame the anaesthetist at all? Dr. Jones comes to the conclusion, in the penultimate paragraph of his paper, that the few minutes saved by the use of a relaxant carries the risk of anoxia and hypercarbia. We should like to point out that one normally hyperventilates adequately with pure oxygen before actual intubation after the administration of a relaxant. There is certainly no time for hypercarbia to develop, and apnoea of at least 4 subsequent minutes will be accompanied by perfect oxygenation by virtue of 'diffusion' respiration. Numerous studies attest to the safety of this, particularly the absence of ECG changes.[7]

As regards the remarks of Dr. Jones about controlled respiration, anyone familiar with the pioneering work done at Liverpool during the last two decades, ably guided by Prof. Cecil Gray, will be convinced, and remain convinced, of its boon to anaesthesia. In this centre over 30,000 infants have now been intubated after the administration of muscle relaxants by house-surgeons, registrars, and consultants, all without any untoward effect [G. Jackson-Rees (1960), personal communication to one of us (L.M.)]. American experience in 3,213 cases is similar.[8] In the final analysis, one must recognize that there is more than one way of 'killing a cat'; there is really no such thing as dangerous methods, or dangerous anaesthetics—only dangerous anaesthetists.

Louis Milner and J. S. I. Pitcher
Department of Anaesthetics
University of the Witwatersrand and Johannesburg Hospital
3 June 1961

1. Jones, C. S. (1961): S. Afr. Med. J., **35**, 421.
2. Watland, D. C., Long, J. P., Pittinger, C. B. and Cullen, S. C. (1957): Anesthesiology, **18**, 883.
3. Denson, J. S. and Joseph, S. I. (1954): *Ibid.*, **15**, 650.
4. Colon-Yordan, E., Mackrell, T. N. and Stone, G. H. (1953): *Ibid.*, **14**, 255.
5. Hampton, L. J., Little, D. M. and Fuller, E. M. (1953): *Ibid.*, **14**, 382.
6. Norris, W. and Stuart, P. (1957): Brit. Med. J., **1**, 860.
7. Rollason, W. N. and Hough, J. M. (1957): Brit. J. Anaesth., **29**, 363.
8. Pender, J. W. (1954): Anesthesiology, **15**, 495.

THE MANAGEMENT OF TRAUMATIC PARAPLEGIC PATIENTS

To the Editor: Allow me to congratulate you on your concise and clear editorial on this subject[1] which was prompted by an article by Mr. T. B. McMurray[2] and a letter by Mr. I. Jacobson.[3] You thought fit to remark that *these contributions served as urgent reminders of the dire need for paraplegic centres in this country* (italics—mine).

To this view I subscribe wholeheartedly and can speak with some experience and feeling on the subject as it concerns the Cape. For a number of years Mr. McMurray and I have taken an interest in the considerable number of paraplegic patients who have collected at the Conradie Hospital, Pinelands, Cape. The majority are males (mostly young adults), of non-European descent, but there are also a few European males and a few females of both races. Most of the cases are the result of trauma—industrial or accidental.

The very fact that these patients are transferred to the chronic section of this hospital designates them permanently disabled with a hopeless prognosis. Many of them arrive as physical wrecks—rotten with sores, emaciated, anaemic, with grossly infected urinary tracts, bedridden with disabling contractures, and with their morale very low. It has been possible to improve the lot of quite a few of these, so that a small band of them strut around the grounds or engage in various activities from their wheelchairs. But all to no gainful purpose. Mr. McMurray correctly emphasized the hopelessness of trying to *manage paraplegics* when their *treatment* and *rehabilitation* is separately vested in Provincial and Central Government authority. Paraplegic patients are a class by themselves and treatment and rehabilitation go hand-in-hand and must be intimately coordinated.

Mr. McMurray has listed a series of essential steps whereby the sorry plight of paraplegic patients in this country can be improved, and I was delighted to read that Mr. Jacobson agreed that the need for paraplegic centres was dire and urgent. Let us make no mistake. We may be justifiably proud of the standard of medicine generally in this country, but in the particular problem of the management of paraplegic patients we must hang our heads in shame. They just don't get a square deal, except for a small group in the Transvaal. As far as the Cape is concerned we have everything that is required—the medical specialities, the nursing staff, the hospital beds, and (if we can illuminate the whole of De Waal Drive), the necessary funds. We lack coordinated authority.

Two basic aims must be kept in mind: (*a*) The paraplegic patient requires to be under medical supervision for the rest of his life, and (*b*) the paraplegic patient needs to be rehabilitated into an occupation which gives him back his self-respect.

Space precludes the opportunity to dilate on these principles, which are in fact self-evident. The medical treatment of paraplegics is quite straightforward. Bedsores, contractures, and loss of morale are all preventable. Debility and urosepsis are treatable. What is required are centres where the paraplegic patient is transferred early in his illness. The aim and object of the centre is first to render the patient medically and physically independent, and then to equip him for his readjusted occupation.

At Stoke Mandeville in Aylesbury (UK) 75% of paraplegic patients return to gainful occupation. Ideally this is done from their own homes, and periodic visits to the centre keeps them medically fit.

In our multiracial country the problem is more formidable, but by no means insoluble. It is being realistically tackled for Bantu patients at Baragwanath Hospital with commendable results. In the Cape the problem is being shelved. This unscientific and inhuman disregard for an increasing problem can no longer be accepted with complacency.

P. J. M. Retief
213 Netherlands Bank Buildings
85 St. George's Street
Cape Town
30 May 1961

1. Editorial (1961): S. Afr. Med. J., **35**, 384.
2. McMurray, T. B. (1961): *Ibid.*, **35**, 1.
3. Correspondence (1961): *Ibid.*, **35**, 100.

THE SOCIETY OF CARDIOLOGICAL TECHNOLOGISTS OF SOUTH AFRICA

To the Editor: Examinations for the Associate Membership of the Society of Cardiological Technologists of South Africa are taking place on 17 June 1961 at Groote Schuur Hospital, Observatory, Cape, under the auspices of the Southern African Cardiac Society.

A further course of instruction for Associate Membership is due to begin in mid-July. The syllabus includes:

1. Fundamentals of electrocardiography.
2. Anatomy and physiology of the heart and circulation.
3. Clinical photography.
4. Principles of sterilization and asepsis.

Those interested should communicate with the Secretary of the Society, not later than 8 July 1961.

It is hoped that in 1962 a course of instruction for Full Membership will begin. The syllabus will include:

1. Principles of physics.
2. Electrocardiography.
3. Electromanometry.
4. Dye-indicator techniques.
5. Blood-gas analysis.
6. Pulmonary functions.
7. Instrumental techniques.

Details regarding this course will be published at a later date in this *Journal*.

L. W. Piller
Chairman
Cardiac Clinic
Groote Schuur Hospital
Observatory, Cape
4 June 1961